VOLUME 50 NUMBER 1

ARCHIVES OF NEUROLOGY AND PSYCHIATRY

JULY 1943

PUBLISHED MONTHLY BY AMERICAN MEDICAL ASSOCIATION, 535 NORTH DEARBORN STREET, CHICAGO 10, ILLINOIS. ANNUAL SUBSCRIPTION, $8.00

Entered as Second Class Matter Jan. 7, 1919, at the Postoffice at Chicago, Under the Act of Congress of March 3, 1879

CONTENTS OF PREVIOUS NUMBER

JUNE 1943. NUMBER 6

Archives of Neurology and Psychiatry

VOLUME 50 JULY 1943 NUMBER 1

INTRAMEDULLARY CAVITATION RESULTING FROM ADHESIVE SPINAL ARACHNOIDITIS

JACK NELSON, M.D.
NEW YORK

In 1897 Schwarz [1] presented before the medical society in Vienna the first clinicopathologic report of a case of chronic adhesive arachnoiditis in which the spinal cord displayed focal areas of softening and cavitation. He expressed the belief that the condition was of syphilitic origin, but the evidence is not available.

Since that time the twin problems of adhesive arachnoiditis and intramedullary cavitation have been the subject of much study, and the question of their interrelation—which is the cause and which the consequence—has received particular attention. Pette,[2] Davison and Keschner,[3] Yasuda [4] and, more recently, Lubin [5] have reported cases presenting a combination of the two features.

The intramedullary cavitation in these and in other instances has often been improperly grouped under the heading of syringomyelia. Even the terms "pseudosyringomyelia" and "secondary syringomyelia" must be considered inappropriate, for they imply an etiologic basis which, if not identical with, is at least similar to that obtaining in syringomyelia. On the contrary, as is indicated both by a study of the literature and by the present case, the pathogenesis of the condition under discussion is basically otherwise.

Syringomyelia, the so-called primary form, is generally considered to be due to a developmental anomaly.[6] The enclosure of the central canal by the process of dorsal overlapping and the filling in of the dorsal columns is faulty, with the result that there remain islands of primitive cell rests consisting of ependymal spongioblasts. This primitive ependymal epithelium proliferates, with resultant surrounding gliosis, interference in the blood supply to the area and eventual necrosis proceeding to cavitation. Hence are derived the basic pathologic characteristics of this condition: its noninflammatory appearance, the close relation of the cavity to the central canal and its possession of an ependymal lining, as well as the considerable gliosis which is commonly seen and the frequent association of the condition with other con-

From the Neurologic Service of Dr. Foster Kennedy, Bellevue Hospital.

1. Schwarz, E.: Präparate von einem Falle syphilitischer Meningomyelitis mit Höhlenbildung im Rückenmarke und besonderen degenerativen Veränderungen der Neuroglia, Wien. klin. Wchnschr. **110**:177, 1897.

2. Pette, H.: Ueber lokalisierte, unter dem Bilde eines raumbeschränkenden Prozesses verlaufende Spinalmeningitis, Arch. f. Psychiat. **74**:631, 1925.

3. Davison, C., and Keschner, M.: Myelitic and Myelopathic Lesions: VI. Cases with a Marked Circulatory Interference and a Picture of Syringomyelia, Arch. Neurol. & Psychiat. **30**:1074 (Nov.) 1933.

4. Yasuda, T.: Zur Frage der Arachnopathia fibrosa cystica proliferans, Deutsche Ztschr. f. Nervenh. **143**:61, 1937.

5. Lubin, A. J.: Adhesive Spinal Arachnoiditis as a Cause of Intramedullary Cavitation, Arch. Neurol. & Psychiat. **44**:409 (Aug.) 1940.

6. Tamaki, K., and Lubin, A. J.: Pathogenesis of Syringomyelia, Arch. Neurol. & Psychiat. **40**:748 (Oct.) 1938.

genital anomalies. Leptomeningitis is occasionally reported in association with syringomyelia, but is rarely present in more than a slight degree and is believed to be entirely derivative. Thickening of the blood vessels with narrowing of their lumens is commonly observed, and its meaning has been variously interpreted.

INTRAMEDULLARY CAVITATION

That intramedullary cavitation may be secondary to other processes in and about the spinal cord is well recognized. Phillipe and Oberthür,[7] despite their use of the term, discussed at some length the distinction between "true syringomyelia" and "pseudosyringomyelia." Many different causes are encountered. A tumor within the cord may break down and cavitate, or sudden hemorrhage into the cord may lead to the same result.[3] Concussion of the cord without hemorrhage, with pronounced edema, may eventuate in necrosis of the tissue with rarefaction. Among infections, syphilitic meningomyelitis is an occasional cause, as is hypertrophic pachymeningitis of the cervical enlargement.[8] A number of authors[9] have described cases of acute encephalomyelitis and meningomyelitis in which rapid demyelination and destruction of tracts have led to the formation of numerous cavities in the cord.

Pathogenesis.—The pathogenesis of the intramedullary cavities in cases of adhesive spinal arachnoiditis is varied, but impairment of the circulation seems to be the mediating mechanism in many of them. In other instances circulatory insufficiency is clearly a more immediate factor. For example, Ornsteen[10] described thrombosis of the anterior spinal artery resulting from relatively minor injuries and indicated that such thrombosis could lead to cavitation within the cord. In a case of Davison and Keschner,[3] marked thickening of the arachnoid on the ventral surface of the cervical enlargement was responsible for compression of the anterior spinal artery and consequent cavity formation.

Unusual cases are sometimes encountered in which an expanding neoplasm of the brain stem is followed by intramedullary cavitation in the lower cervical segments of the cord. In such a case, reported by Harbitz and Lossius,[11] an epithelioma on the dorsal aspect of the medulla caused death by compression. Autopsy revealed, besides the epithelioma, diffuse arachnoiditis and a syringoid cavity within the cervical portion of the cord. In a similar instance described by Tauber and Langworthy,[12] a glioma of the midbrain and pons was complicated by a cavity 7 cm. below the level of the tumor, in the cervical portion of the cord. The mechanism of this interesting condition is believed to be as follows: Just before the vertebral arteries unite at the medulla to form the ascending basilar artery, they give off two branches, which descend and unite to form the anteromedian spinal artery. This vessel

7. Phillipe, C., and Oberthür, J.: Classification des cavités pathologiques intra-médullaires: I. Syringomyélie vraie; forme cavitaire et forme pachyméningitique; II. Pseudo-syringomyélies (hydromyélie; hématomyélie; cavités vasculaires; cavités traumatiques), Rev. neurol. **8**:171, 1900.

8. (*a*) Kennedy, F., and Holmes, G.: Two Anomalous Cases of Syringomyelia, Proc. Roy. Soc. Med. **2**:1, 1908. (*b*) Denker, P. G., and Kennedy, F.: Congenital Syphilitic Syringomyelia with Arthropathy of Elbow, J. A. M. A. **114**:408 (Feb. 8) 1940.

9. Biemond, A.: Ueber die Meningo-radiculo-neuritis (Guillain-Barré) und die Meningomyelo-encephalitis, betrachtet als Krankheiten bei denen Agens primär in Meningealraum angreift, Deutsche Ztschr. f. Nervenh. **143**:172, 1937. Martin, J. P.: Amyotrophic Meningomyelitis, Brain **48**:153, 1925.

10. Ornsteen, A. M.: Thrombosis of the Anterior Spinal Artery, Am. J. M. Sc. **181**:654, 1931.

11. Harbitz, F., and Lossius, E.: Extra-Medullary Tumor: Arachnitis Fibrosa Cystica et Ossificans; Gliosis of the Medulla, Acta psychiat. et neurol. **4**:51, 1929.

12. Tauber, E. S., and Langworthy, O. R.: A Study of Syringomyelia and Formation of Cavities in the Spinal Cord, J. Nerv. & Ment. Dis. **81**:245, 1935.

extends downward without collateral circulation until, at the level of the sixth cervical segment, it is joined by segmental arteries. It is thought that the formation of a pressure cone at the foramen magnum causes narrowing of the anteromedian spinal artery, with resultant ischemic necrosis of that section of the cervical part of the cord, which, lacking segmental arteries, is particularly vulnerable.

Tauber and Langworthy,[12] investigating the effect of vascular occlusion at various levels of the spinal cord, observed that compression of the ventral half of the cord, including the anterior blood vessels, in cats produced fusiform cavities situated at the base of the posterior columns and extending over several segments. Whether a diminution of the arterial supply or of the venous drainage is basically responsible for intramedullary cavitations of most cases is difficult to say, but it seems likely that impairment of the circulation in some manner is a major factor in the formation of "secondary" intramedullary cavitation.

ADHESIVE ARACHNOIDITIS AND INTRAMEDULLARY CAVITATION

The close relation of adhesive arachnoiditis to intramedullary cavitation was manifest in the original case of Schwarz and has repeatedly been affirmed since. In that report, and for many years afterward, syphilis was looked on as the almost invariable cause of such arachnoiditis.[13] But in 1919 Mauss and Krüger,[14] in a review of war injuries of the spinal cord, observed adhesive arachnoiditis as an occasional sequela of such injury. Until lately the condition was rarely diagnosed before operation, being confused with such disorders as multiple sclerosis and lateral sclerosis, and it received attention chiefly in connection with the circumscribed cysts of the arachnoid which simulated tumors of the cord.[15]

In recent years, however, it has been recognized that adhesive arachnoiditis may result from various processes. Bagley,[16] in 1928, injected the blood of dogs into the animal's subarachnoid space by way of the cisterna magna and demonstrated a frequent meningeal reaction in response to a few cubic centimeters of blood. In most instances this reaction progressed to proliferative meningitis and occasionally eventuated in fibrotic organization. In a subsequent report Bagley[17] cited clinical cases of a similar nature. More recently, Merwath and Freiman[18] described the occurrence of adhesive arachnoiditis following spontaneous subarachnoid hemorrhage in an infant.

Mackay[19] pointed out that adhesive arachnoiditis is a stereotyped reaction on the part of the meninges. He noted it after infectious, especially purulent, meningitis

---

13. Horsley, V.: A Clinical Lecture on Chronic Spinal Meningitis: Its Differential Diagnosis and Surgical Treatment, Brit. M. J. **1**:513, 1909.

14. Mauss, T., and Krüger, H.: Ueber die unter dem Bilde der Meningitis serosa circumscripta verlaufenden Kriegsschädigungen des Rückenmarkes und ihr operative Behandlung, Deutsche Ztschr. f. Nervenh. **62**:1, 1919.

15. Spiller, W. G.; Musser, J. H., and Martin, E.: A Case of Intradural Spinal Cyst with Operation and Recovery, Univ. Pennsylvania M. Bull. **16**:27 and 56, 1903-1904. Spiller, W. G.: Circumscribed Serous Spinal Meningitis: A Little Recognized Condition Amenable to Surgical Therapy, Am. J. M. Sc. **137**:95, 1909. Elkington, J. St. C.: Meningitis Serosa Circumscripta Spinalis, Brain **59**:181, 1936.

16. Bagley, C., Jr.: Blood in the Cerebrospinal Fluid: A. Experimental Data, Arch. Surg. **17**:18 (July) 1928.

17. Bagley, C., Jr.: Blood in the Cerebrospinal Fluid: B. Clinical Data, Arch. Surg. **17**:39 (July) 1928.

18. Merwath, H. R., and Freiman, I. S.: Hydrocephalus Following Subarachnoid Hemorrhage, Brooklyn Hosp. J. **1**:149, 1939.

19. Mackay, R. P.: Chronic Adhesive Spinal Arachnoiditis, J. A. M. A. **112**:802 (March 4) 1939.

at periods varying from three months to seventeen years. Barker and Ford [20] reported a case following lymphocytic choriomeningitis. Many authors, among them Doyle,[21] have emphasized that it may be consequent on a low grade chronic meningitis, which may be of subclinical intensity or may otherwise escape clinical recognition.

Of late, spinal arachnoiditis has assumed increasing importance in the neurologic service at Bellevue Hospital, largely as a result of Pool's [22] studies with intraspinal endoscopy (myeloscopy). This method often allows the early diagnosis of the condition and has shown it to be of more frequent occurrence than had previously been recognized.

Adhesive arachnoiditis from any of these causes may, if of sufficient degree, lead to changes in the cord. The precise relation is often unclear, for in most instances, as with purulent meningitis, the occurrence of simultaneous myelitis cannot be ruled out, and in arachnoiditis following trauma the possibility of simultaneous injury to the cord likewise exists. The mechanism in the production of cavities, as has already been indicated, is probably circulatory impairment. Simple compression of the cord, such as might result from the contraction of fibrous tissue, or as is often seen with extramedullary tumor, does not cause intramedullary cavitation. Indeed, cases have been reported by Keschner, Davison and Selinsky [23] in which such a tumor was present without evidence of cavitation of the cord and chronic adhesive arachnoidal process, with consequent cavity formation, followed its removal. Both Mackay [19] in the paper previously referred to, and Lubin,[5] in his recent report of a similar case, expressed the opinion that the adhesive process led to vascular changes, including perivascular cuffing and obliterative thickening of the walls, which they asserted was the immediate cause of the changes in the cord. Stevenson [24] has indicated his belief that the venous drainage from the cord may be of greater importance in cavity formation than the vascular supply to the cord.

REPORT OF A CASE

I present a case of adhesive arachnoiditis of the spinal cord with intramedullary cavitation which possesses several points of unusual interest: First, the sequence of events can be clearly ascertained, the patient having been under observation (*a*) prior to any evidence of meningeal involvement, (*b*) subsequently, when there was evidence of adhesive meningeal process, confirmed by myeloscopic examination, but before signs of intramedullary disease existed, and (*c*) finally when clinical evidence of intramedullary cavitation was present, confirmed by autopsy. Second, a probable etiologic factor for the entire process can be adduced.

J. S., aged 50, was admitted to the neurologic service on May 21, 1939.

The history was long and involved a number of factors of possible etiologic significance. As a child the patient had frequent nosebleeds and bruised easily; these symptoms disappeared at the age of 26 years. In January 1936, when the patient was 46, severe spontaneous nosebleeds recurred, followed about a month later by the onset of frequent convulsive seizures. In September of the same year he was admitted to the Bellevue Psychiatric Pavilion, where he showed irritability and gross memory defect, with confabulation and disorientation.

20. Barker, L. F., and Ford, F. R.: Chronic Arachnoiditis Obliterating the Spinal Subarachnoid Space, J. A. M. A. **109**:785 (Sept. 4) 1937.

21. Doyle, J. B., in discussion on Mackay.[19]

22. Pool, J. L.: I. Direct Visualization of the Dorsal Nerve Roots of the Cauda Equina by Means of a Myeloscope, Arch. Neurol. & Psychiat. **38**:1308 (June) 1938; II. Myeloscopy: Diagnostic Inspection of the Cauda Equina by Means of an Endoscope (Myeloscope), Bull. Neurol. Inst. New York **7**:178, 1938; III. Myeloscopy: Intraspinal Endoscopy, Surgery **11**:169, 1942.

23. Keschner, M.; Davison, C., and Selinsky, J., cited by Doyle.[21]

24. Stevenson, L.: Personal communication to the author.

There were many spontaneous ecchymoses on the legs. Studies of the blood revealed as outstanding changes: thrombopenia, with a count of 20,000 platelets, and prolongation of the bleeding time to twenty minutes. Except for mild anemia, the blood picture was normal. Determination of the clotting time, the fragility test, and chemical studies of the blood gave normal results. The Wassermann reaction of the blood was negative. A diagnosis of thrombopenic purpura was made, and after radiation therapy had been given without effect, splenectomy was performed. Microscopic examination of the spleen showed "an excess of eosinophils, highly suggestive of thrombopenic purpura, together with an excess of pigment, such as is present in cases of long-standing malaria." Though the platelet count, after a short increase, returned to a low level, the bleeding time became normal.

Two months later, while the patient was still in the hospital, he had another generalized convulsion. A lumbar puncture, performed the following day, elicited a free flow of spinal fluid, which, however, was bloody and xanthochromic. Subsequently, after his discharge from the hospital, he was followed in the hematology clinic; during this period he had several admissions to the psychiatric division for alcoholism.

In May 1939 the patient was referred to the medical service for convulsions and a post-epileptic clouded state. Examination again revealed evidence of thrombopenic purpura. Neurologic study disclosed some weakness of the left hand and spasticity and circumduction in use of the left leg, with ataxia of this extremity; the reflexes were increased on the left side; the abdominal reflexes were absent bilaterally, and Babinski and Rossolimo signs were present on the left side more than on the right. Vibration sense was diminished over the feet, but no other sensory abnormality was found. Lumbar puncture, attempted at each interspace from the first to the fourth lumbar segment, revealed initial pressure readings of zero and complete manometric block; not more than 4 to 6 cc. of fluid, which was xanthochromic, could be withdrawn. The Wassermann reaction of the spinal fluid was negative, and the colloidal gold curve was normal.

Attempts to obtain an air myelogram were unsuccessful, for only 5 cc. of fluid could be obtained and injection of 3 cc. of air caused positive pressure and much pain. Myeloscopic examination (Dr. Pool) at the second lumbar level revealed that the dura was taut and the spinal nerve roots immobile, as though fixed by adhesions, but only a few scant adhesions were seen.

The patient was discharged with the diagnosis of (1) thrombopenic purpura, which was thought to be responsible for a local hemorrhage in the right cerebral hemisphere, with concomitant convulsive disorder and left hemiparesis, and (2) spinal adhesive arachnoiditis in the lumbar portion of the cord.

In October 1940, four years after his first admission to the hospital, and fifteen months after the second admission, the patient was readmitted to the hospital with the complaint of pains and increasing weakness in both legs for three months. The appearance of these symptoms was followed by constipation and retention of urine passing over into dribbling, incontinence and loss of power of erection. Examination revealed flaccid paresis and wasting of the lower extremities, but no fibrillations were observed. The reflexes were more pronounced on the left side, with diminution of knee jerks and absence of ankle jerks, and the plantar responses were pathologic bilaterally. Sensory testing was difficult, but revealed a band of hypalgesia and hypesthesia from the ninth thoracic to the first lumbar segment; sensibility over the legs seemed normal, but the saddle area (from the second to the fifth sacral segment) was also involved. Vibration sense was absent below the hips, and figure writing was impaired, but position sense was good. The bladder was distended and atonic, and rectal tone was lacking; there were decubital ulcers over the sacrum. The diagnosis was similar to that on the previous admission; in addition, there were recent pachymeningitis of the spinal cord at the lumbar level, with intramedullary disease in the lumbar portion of the cord and the conus, and meningitic involvement of the cauda equina.

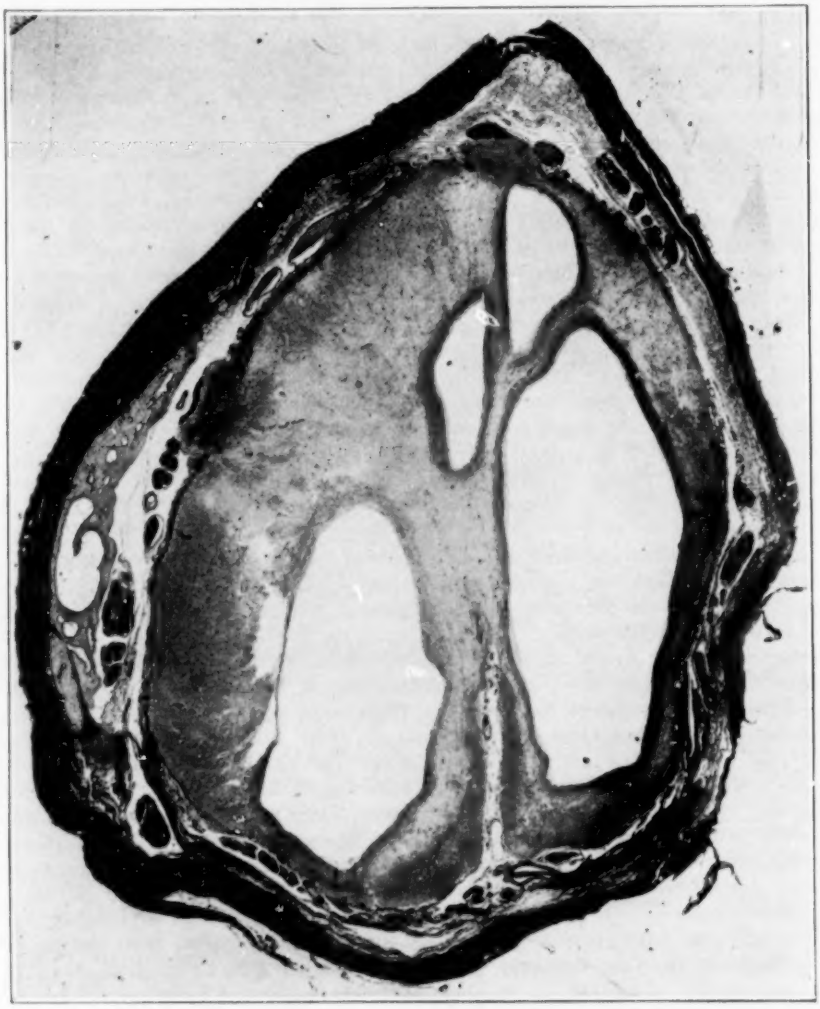
Lumbar punctures were done from the twelfth dorsal to the third lumbar level, but no fluid could be obtained at any point. An inlying catheter with tidal drainage was set up, but the patient's condition steadily declined. Generalized urinary infection, complicated by hematuria, led to renal insufficiency with uremia, and the patient died on the thirty-fifth day of hospitalization.

Autopsy.—General Observations: The outstanding changes were related to the urinary tract. A marked degree of cystopyelonephritis was present. The bladder wall, further, had an area of gangrene, which was sealed against the pelvic mesentery and was surrounded by an area of local peritonitis. The bone marrow was diffusely hypoplastic. There were a mild degree of coronary sclerosis and a small area of ulceration in the lower third of the esophagus.

Central Nervous System (Dr. Lewis D. Stevenson): Brain: The dura was adherent to the brain in numerous places over the cortex. The pia-arachnoid over the frontal region was

thickened to present a milky appearance. Adhesions were noted around the optic nerves, over the temporal lobes and around the cerebellum and pons. Moderate cortical atrophy and some basilar arteriosclerosis were present. In the midregion of the right hemisphere, about 3 cm. beneath the cortex, and outside the internal capsule, there was an area of grayish discoloration and softening. Microscopic examination of this area revealed perivascular and perineuronal edema, with pyknosis and chromatolysis of cells and collections of fat around the blood vessels.

Spinal Cord: Beginning at about the level of the midthoracic region, the dura became greatly thickened and adherent to the cord in its entire circumference. This process extended to the termination of the cauda equina. In the region of the cauda equina numerous strand-



Cross section of the spinal cord at the lumbar level, showing pronounced thickening of both the dura and the arachnoid, with almost complete obliteration of the subarachnoid space. Subdurally to the left lies a calcified plaque. The cord itself contains a number of cavities, which occupy most of its cross section, particularly in the area of the gray matter.

like adhesions were attached to the greatly thickened dura, and the nerves were adherent to one another. Section of the cervical portion of the cord showed normal architecture and no cavities, but section of the lower dorsal part of the cord revealed several cavities and the architecture appeared to be entirely lost. In the lumbar region of the cord there was a large central cavity, in contrast to the several cavities that were present in the higher region.

Microscopic Observations.—The dura was somewhat thickened in places. There was also notable thickening of the arachnoid, which was closely adherent to the dura and to the sur-

face of the cord in many areas, with considerable obliteration of the subarachnoid space. The blood vessels in the subarachnoid space appeared normal. At one point in the arachnoid occurred a calcified plaque, containing many spaces. Some of these spaces were filled with fat, and others contained fat, together with large numbers of phagocytes. In the plaque were also a number of blood vessels. The nerve roots seemed to lie fairly free in spaces in the arachnoid. The cord itself contained two large cavities and two smaller cavities which occupied most of its cross section. These cavities were surrounded, particularly in the anterolateral regions of the cord, by a narrow rim of fairly normal white substance. The arterioles within this white substance showed a slight degree of sclerosis. The cavities occupied the position of most of the gray matter, only remnants of which survived, in the form of a few degenerated anterior horn cells in the necrotic tissue about the two larger cavities. Remnants of the central canal in the form of ependymal cells were observed near the center of the spinal cord. No ependymal cells could be seen lining any of the cavities. The condition seems to be secondary syringomyelia, associated with chronic adhesive arachnoiditis and without evidence of recent inflammation.

COMMENT

The picture is, therefore, one of a long-standing proliferative process which involved all the meninges and led to adhesive arachnoiditis, indeed, to panmeningitis, extending over the brain and the spinal cord. In the cord the condition was sufficiently severe to interfere with the nutrition of the cord. The intramedullary cavitation may be presumed to have been secondary to this process; the mechanism, though not altogether clear, must have consisted in part of circulatory impairment.

In this case the question of the etiologic factors is peculiarly interesting. Adhesive arachnoiditis represents the end stage of a process the cause of which is often indeterminable; it is a stereotyped reaction to many possible causes. In this case there was no evidence of syphilis and no history of infectious meningitis. The onset of symptoms was gradual, unlike that associated with purpuric hematomyelia. Further, evidence of complete block, with obliteration of the subarachnoid space, was noted long before the onset of symptoms of intramedullary disease. The patient fell into the age group in which such a process is sometimes called "senile arachnoiditis," but this term furnishes no hint of an etiologic factor. The history of chronic alcoholism with malnutrition might possibly bear a relation to the disease, but this is doubtful.

The patient, however, had thrombopenic purpura, and during at least one of his many convulsions, meningeal bleeding is known to have occurred. It appears likely that such bleeding, occurring repeatedly, as it did during his seizures, may have initiated sterile meningitis, which proceeded to fibrotic organization. The experiments of Bagley and the clinical reports of Bagley and of Merwath and Freiman have already been referred to in this connection. The cavitation within the cord was probably secondary to the adhesive process in the meninges.

SUMMARY

A case of intramedullary cavitation of the spinal cord secondary to adhesive arachnoiditis is presented. A possible etiologic factor in this case is indicated.

535 Park Avenue.

EFFECT OF METRAZOL CONVULSIONS ON CONDITIONED REFLEXES IN DOGS

VICTOR H. ROSEN, M.D.
AND
W. HORSLEY GANTT, M.D.
BALTIMORE

The whole question of shock therapy has been surrounded with confusion. Not only is the action nonspecific, but there are reports on the one hand of decided improvement and, on the other, of histologic damage to the tissues of the brain, including the cerebral cortex, as well as gross anatomic evidence of injury to bones. But a certain amount of damage to the brain may occur without loss of important functions. Discussions to date concerning the reasons for alterations in behavior following shock therapy have dealt with changes in cerebral circulation, the chemistry and p_H of the body fluids and ion changes in the cell membrane, histologic changes in the cerebral cortex, changes in autonomic and hormone balance, fear of the therapy, satisfaction of need for punishment and the death urge and changes in memory. As few controlled experiments had been undertaken to determine what alteration may occur in the higher integrated activity of the nervous system, and as it was simpler at first to make such a study with animals, we investigated the effect of metrazol convulsions on the behavior of dogs, chiefly by the conditioned reflex method.

The conditioned reflexes were chosen for study because they represent mentally integrated, measurable responses, which under certain controlled situations can be predicted with a high degree of certainty so that their alteration or failure to appear can be ascribed to new variables that have been brought into the rigidly controlled setup. The importance of the study of the effect of shock treatment on the mentally integrated symbolic functions of the total organism is obvious, though it should be emphasized that this is only one of the many possible approaches to this problem.

METHOD

Four adult dogs were used in our experiments. Two were animals in which salivary fistulas had previously been prepared and in which the salivary responses to stimuli reenforced by food were studied, and 2 were animals in which the conditioned defense reactions to an electrical shock to the forepaw were observed. The conditioned stimuli used were auditory tones or metronome beats of various frequencies. Two types of responses were chiefly studied. A crude conditioned response was formed to one sound, and differential conditioned responses were formed to two metronomes beating at different, but progressively closer, frequencies, the one beat (hereafter designated as the positive stimulus) being reenforced by the unconditioned stimulus and the other (hereafter designated as the negative stimulus) not being so reenforced. The following data were recorded during the procedure: the general behavior of the animal, the time and character of the orienting reflex (the animal's getting his body into position, so that one sees he is turning toward the stimulus; in other words, there are sharp turning of the head, focusing of the eyes on that spot and pricking up the ears, giving the impression that the animal is paying more attention to the stimulus than to anything else), the latent period of the conditioned response, the amplitude of the

From the Pavlovian Laboratory, the Phipps Psychiatric Clinic, Johns Hopkins University.
Read at the Sixty-Eighth Annual Meeting of the American Neurological Association, June 4, 1942.

conditioned response, the amplitude of the unconditioned response and the accompanying conditioned respiratory and cardiac rates. In the motor response experiments kymographic records were taken of the actual foot-lifting defense reactions, together with cardiac and respiratory rates.

After a sufficient training period and a control period, during which a fair constancy of the conditioned responses was attained, the course of metrazol convulsions was begun. The drug was given three times a week on alternate days until a series of ten to twelve convulsions had been induced. The convulsive pattern in the dog, the latent period of the convulsion, the length of the tonic and the clonic phase and the total period of apnea were recorded. The patterns of the convulsions were remarkably similar from day to day and from dog to dog. The general behavior of all 4 dogs may be described as follows: After the intravenous injection of the drug, there was a tonic spasm, with a latent period of six to fourteen seconds, during which the dog would fall suddenly to the floor, with wide-opened mouth, retraction of the head and flexion of the paws and usually involuntary micturition. This stage lasted from ten to fifteen seconds and was followed by violent jerking, clonic movements of the body, legs and jaws, profuse salivation, cyanosis of the tongue and dilatation of the pupils. This stage usually lasted about forty-five to fifty seconds and was terminated by relaxation of the body and a deep sigh. The total period of apnea averaged slightly less than a minute. After the first breath there were usually active running movements of all four legs, associated with struggling attempts to achieve the erect posture. Finally, after several minutes of this behavior, the animal usually staggered to his feet and stood swaying for several minutes more, dazed and paying no attention to ordinary stimuli. The first of the more highly integrated reactions to reappear, in about fifteen to twenty minutes, were the orienting reflex and defense movements. Food was usually refused for several hours after the seizure. When the dog was fully recovered from the convulsion, he would stretch out on the floor, all spontaneous activity reduced to a minimum.

During the course of convulsions the dog became thinner and less active. Appetite appeared to increase greatly on the interval days. There was less playfulness, and most of the dogs seemed to be less sure of their destination when en route from the cage to the laboratory; previously they had led the way and stopped at the right door, but after the convulsion they appeared hesitant and ran past the door. Two of the dogs showed some apprehension and resistance to the injections, but the other 2 animals gave no evidence of anticipating any unpleasant reaction when approached with the needle and syringe. A salivary secretion in response to the smell of the metrazol was noted in all 4 dogs, as well as a tendency to brief periods of convulsive twitching for many hours after the seizure, and even on the interval day, when the dog was moving about his cage.

The motor dogs were tested during the postconvulsive period (about twenty minutes to one-half hour after recovery) and on the interval day with the same routine of stimuli that had been used during the training period. The salivary dogs were tested only on the interval days, since the profuse salivation and the lack of appetite made routine postconvulsive testing impossible; it was evident, however, that both conditioned and unconditioned food reflexes were in abeyance. After the series of convulsions had been terminated, the dogs were tested at weekly intervals during the period of recovery. Hereafter, "postconvulsive period" means the period following the convulsion when the dog first becomes conscious and is able to walk about normally; "interval day" signifies the day between seizures, and "recovery period" indicates the period following cessation of the course of metrazol seizures.

Salivary responses were measured in millimeters of water; heart rates were recorded electrically with subcutaneous needle electrodes, and respiratory patterns, with the Manning pneumograph.

RESULTS

Dog 1.—Peik, about 6 years of age and weighing 19.4 Kg., was a strong, active dog with a prepared salivary fistula; he was a moderately stable animal, which had been in the laboratory since 1939 and had formed positive and negative conditioned reflexes. In the present study, he was trained to a point at which he gave a fairly large and constant secretion to a food-conditioned metronome stimulus with a frequency of 100 per minute (M 100 +) and no secretion to a metronome stimulus with a frequency of 80 per minute (M 80 —) which was not followed by food. Thus, he showed an adequate differentiation of positive and negative stimuli. A constant secretion was also given as a crude conditioned response to a bubbling noise (Bu +). A series of ten metrazol seizures were then induced, as already described.

Figure 1 shows the changes brought about in the quantitative values of the salivary conditioned reflexes in Peik. After the fifth and sixth seizures a sharp rise in the reaction to the negative stimulus (M 80 —) was noted, from zero to 250 mm.; there was no longer any differentiation between the positive and the negative metronome stimulus. After the eighth convulsion all responses fell precipitately to zero, where they remained until about two weeks after the last convulsion, when they reappeared in reverse order. The first

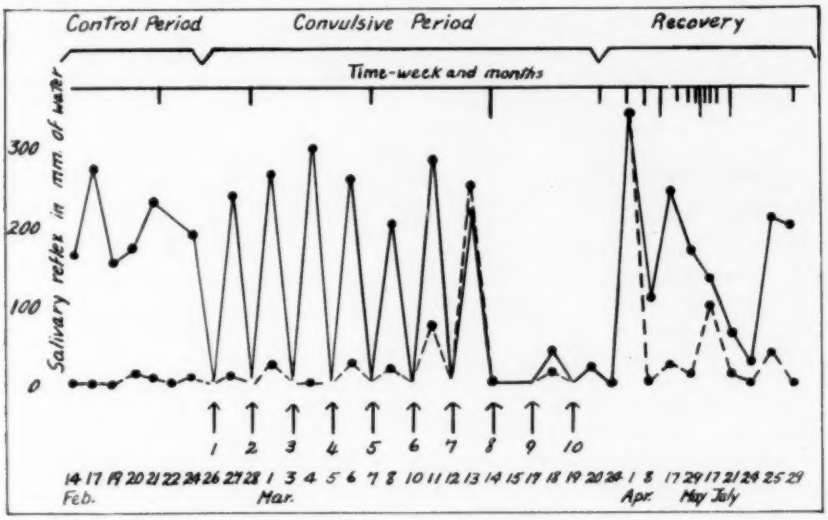


Fig. 1 (Peik).—Effect of metrazol convulsions on the conditioned salivary reflexes. Note the loss of differentiation of stimuli after seven convulsions, the loss lasting four months. No measurement of conditioned reflexes was made on convulsion days. The upper solid line indicates excitatory responses; the lower, dotted line, inhibitory responses. Arrows indicate occurrence of convulsions. Time is expressed in weeks. Compare with figures 2 and 3.

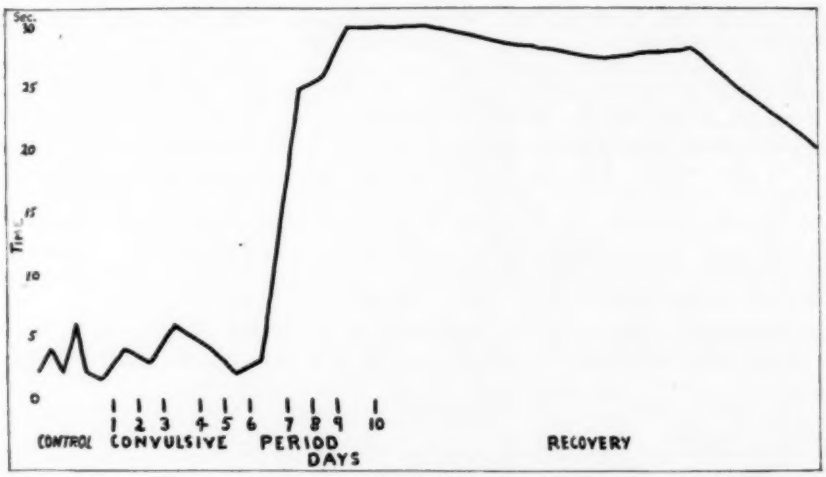


Fig. 2 (Peik).—Effect of metrazol convulsions on the orienting reflex response to a negative stimulus sounding for thirty seconds. Compare with figure 1. Note that the appearance of the orienting reflex coincides with the loss of ability to differentiate between stimuli, both appearing after the seventh convulsion.

response at this time showed lack of differentiation; the responses to both the negative and the positive stimulus were far above those of the control period. The response to the negative metronome stimulus exceeded the response to the positive stimulus. Within three weeks there was a spontaneous return of the differentiation to levels noted in the control period.

Retraining during the postconvulsive period was not attempted; the routine was exactly the same but was carried on once a week instead of every day, as in the training period.

Figure 2 shows the effect of the convulsions on the orienting reflex. During the control period the dog usually oriented to the negative metronome stimulus (M 80—) for three to four seconds at the beginning of the thirty second period during which it sounded. After this brief turning of the head and pricking up of the ears, he would hang his head and appear drowsy for the rest of the period. After the seventh and eighth seizures there was a sudden sharp increase in the length of the orienting period, the dog remaining alert and with ears and eyes turned toward the stimulus until it ceased to sound. This prolongation of the orienting time began to drop off only after six weeks of the recovery period.

An additional phenomenon, designated as the "after-secretion," or delayed excitatory response, also made its appearance after the seventh and eighth convulsions (fig. 3). During the control period the amount of spontaneous salivary secretion in the intervals between the conditioned stimuli averaged 30 to 35 mm. After the seventh convulsion (fig. 3) a large after-secretion amounting to about 200 mm. in one minute, was noted, beginning about ten seconds after the cessation of the thirty second exposure to the negative metronome stimulus (M 80—). The curve for this response followed closely that for the orienting reflex, both phenomena appearing after the seventh convulsion, at which time the dog had also lost his ability to differentiate between the positive and the negative metronome stimulus. This phenomenon was probably due to weakening of inhibition, i. e., disinhibition.

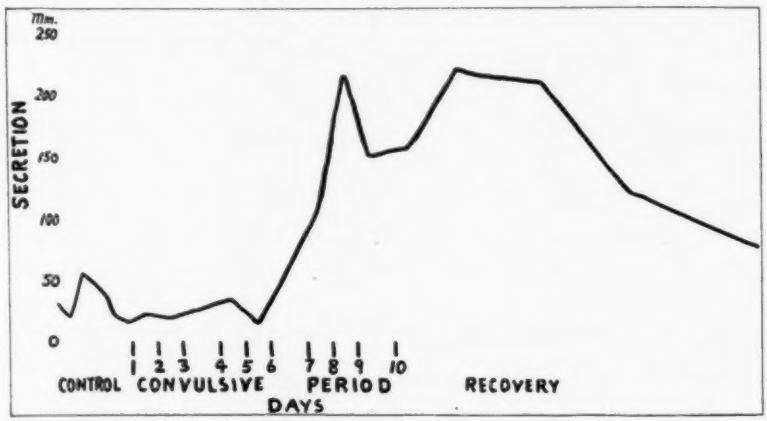


Fig. 3 (Peik).—Effect of metrazol convulsions shown in the spontaneous "after-secretion," appearing immediately after the cessation of a thirty second negative stimulus. The phenomenon illustrates removal of inhibition, the equivalent of delayed disinhibition. Compare with figures 1 and 2.

Dog 2.—Sechs, a stable police dog about 8 years old, had been used in the laboratory for the production of food-conditioned reflexes since 1936, when a salivary fistula had been made.

Before the metrazol experiments Sechs had made an extremely stable differentiation between the positive stimulus (M 100 +) and the negative stimulus (M 80—). Besides the measurable secretion, there was also a differentiation in the cardiac rates accompanying the responses to these stimuli.

Ten metrazol convulsions were given between April 25 and May 22, 1941. Sechs showed less impairment than did the other dogs. Figure 4 shows, however, that there was a fall in the positive conditioned reflex to about 50 per cent of its control value, as well as a rise in the inhibitory conditioned reflex, i. e., a conversion of the inhibitory into an excitatory response, with consequent loss of differentiation. This effect on the excitatory conditioned reflex was more pronounced with a weak excitatory stimulus, represented by a light (L 40 +), than it was with the stable excitatory stimulus (M 100 +). A conspicuous feature of Sechs's behavior, as well as of Connie's, was the length of time necessary for recovery: The response to the weak excitatory stimulus (L 40 +) gradually sank to zero and remained so until the end of July 1941; at the same time the response to the strong excitatory stimulus (M 100 +) continued to be less than 50 per cent of its control value. No after-effect was seen on the response to the inhibitory stimulus (M 80—). After an intensive period of retraining, begin-

ning on November 5, the response to an excitatory stimulus (M 100 +) gradually became restored to its former control value. The cardiac conditioned reflexes showed about the same differentiation as did the salivary responses.

Dog 3.—Motor dog "Connie" was a stable police dog about 3 years old. Brought into the laboratory in November 1940, by February 1941 he was able to make a stable differentiation between a positive stimulus of 92 a minute (M 92 +) and a negative stimulus of 100 a minute (M 100 —). The dog, however, failed to differentiate between a positive stimulus of 96 a minute (M 96 +) and a negative stimulus of 100 a minute (M 100 —). In April 1941 ten metrazol convulsions were given. There was complete loss of the differentiation between stimulus M 92 + and stimulus M 100 —, not only throughout the convulsive period but until

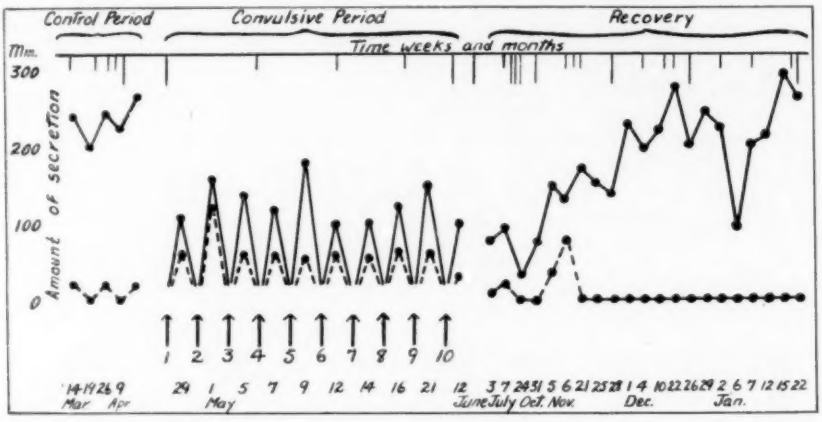


Fig. 4.—Effect of metrazol convulsions on the conditioned salivary reflexes in Sechs. Note the decreased excitation and loss of inhibition (impaired differentiation), which lasted about seven months. Excitatory conditioned reflexes are indicated by a solid line; inhibitory ones, by a dotted line. Time is marked in weeks (short lines) and in months (long lines).

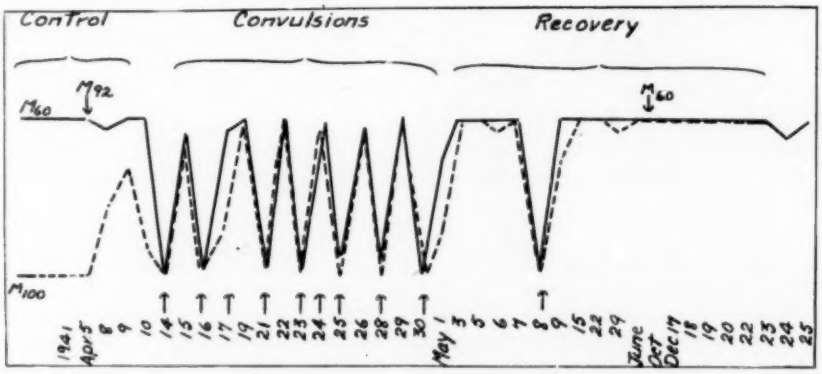


Fig. 5.—Effect of metrazol convulsions on the motor conditioned responses of a stable dog (Connie). Note the complete loss of conditioned responses on convulsion days and the loss of inhibition on interval days (impaired differentiation), which lasted more than eight months.

The solid line indicates the response to the positive metronome stimuli of 92 and 60 beats a minute (M 92 + and M 60 +); the broken line, the responses to the negative stimulus of 100 beats a minute (M 100 —). Arrows indicate injections of metrazol.

the end of October. This loss persisted even though differentiation was made easier by the use of two metronome beats of frequencies farther apart. Ten test training periods were given after the last convulsion; this number would not have been sufficient for thorough retraining, but some differentiation should have resulted from the training, as the animal, previous to any convulsions, had made a nearly perfect differentiation after eight training days.

On the convulsion days the responses to both conditioned reflexes fell to zero, while on the interval days and in the postconvulsive period they both became excitatory.

On Dec. 17, 1941, Connie was given a new period of training to differentiate between a tone of middle C and its octave (T 256 + and T 512 —). There was no differentiation after six training days, in contrast to the excellent differentiation he had made in December 1940 (fig. 5). However, by Jan. 10, 1942 Connie had apparently returned to his former ability to differentiate, and since that time he has remained stable. It is of interest that subjection of this dog, as well as of Sechs, to anoxia corresponding to that occurring at 25,000 feet (7.6 kilometers) for four hours produced only a slight and temporary loss of conditioned reflex function, in contrast to the loss produced by metrazol convulsions.

Dog 4.—In contrast to the preceding 3 stable animals, Laval was a restless, hyperactive animal, a small, wiry, demonstrative, brindle and white mongrel bull terrier, about 1 year of age and weighing 9 Kg. His conditioned reflex training began ten weeks before the course of metrazol was given. The animal was very excitable and in his motor responses differentiated poorly between a positive metronome beat of 100 a minute (M 100 +), followed by a shock to the left forepaw in three seconds, and a negative metronome beat of 40 a minute (M 40 —) for three seconds, which was not followed by a shock. When the negative metronome stimulus was increased to a frequency of 60 a minute (M 60 —), there was indiscriminate foot lifting to both the positive and the negative metronome stimulus.

However, there was some qualitative, if not quantitative, evidence that the dog differentiated to some extent between the two metronome stimuli. His foot lifting was less violent to stimulus M 60 —, and he returned his foot to the metal plate before the metronome had stopped beating more frequently with stimulus M 60 — than with stimulus M 100 +.

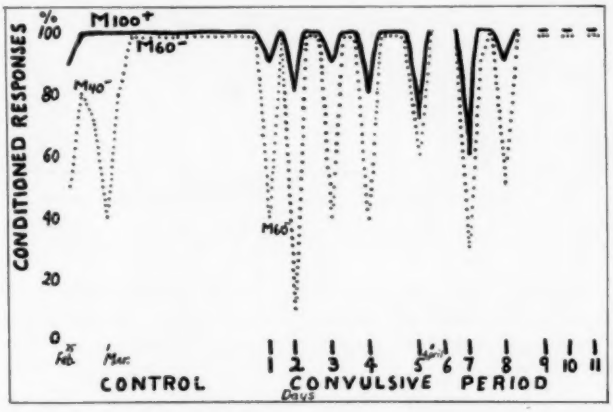


Fig. 6.—Effect of metrazol convulsions on motor conditioned responses of a labile, hyperactive dog (Laval). Note the improved differentiation, due to decreased excitation. See figure 7.

Eleven metrazol convulsions were administered, with the following changes: There was pronounced loss of weight; severe mange developed during the course of the convulsive therapy; the animal became less active and much less demonstrative; after about the sixth convulsion he lost interest in the experimenter, whom he had previously eagerly awaited, and he began to lose his way in going from his cage to the experimental camera.

This animal was tested both shortly after the convulsive seizure and on the interval days. Figure 6 shows the effect of the convulsion on the ability to differentiate the two metronome beats. It can be seen that there is apparent improvement in differentiation, with fewer responses to the negative metronome stimulus and, moreover, though to a lesser degree, fewer responses to the positive metronome stimulus. On the interval days the excitatory behavior is seen to reappear. This phenomenon shows an interesting correlation with the foot-lifting responses (fig. 7). Record *A* represents the preconvulsive period; it can be seen that there is rapid multiple lifting of the left forepaw to both the positive and the negative metronome stimulus, with many random movements between the stimuli. Record *B*, following immediately on recovery from the convulsion, on the same day, shows a single lift of the paw to the positive metronome stimulus, no response to the negative metronome stimulus and dropping out of random movements. Record *C* is the record of an interval day after the ninth convulsion. There is, again, no real differentiation, but there is still a marked decrease in random movements. Figure 8 shows the latent period of the conditioned defense reaction of the left forepaw after the convulsion and on the interval days. In the postconvulsive period there was noticeable prolongation of the latent period. The animal not only differentiated

more efficiently but waited almost until the moment of the shock before removing the left paw from the metal plate. Thus, the whole performance was more economical than before the metrazol convulsions were produced in this excitable animal.

In all the dogs the sexual unconditioned reflexes were abolished on the days on which the convulsions were produced, but they returned to normal on the interval days. The unconditioned salivary reflexes, likewise, showed little, or no, alteration.

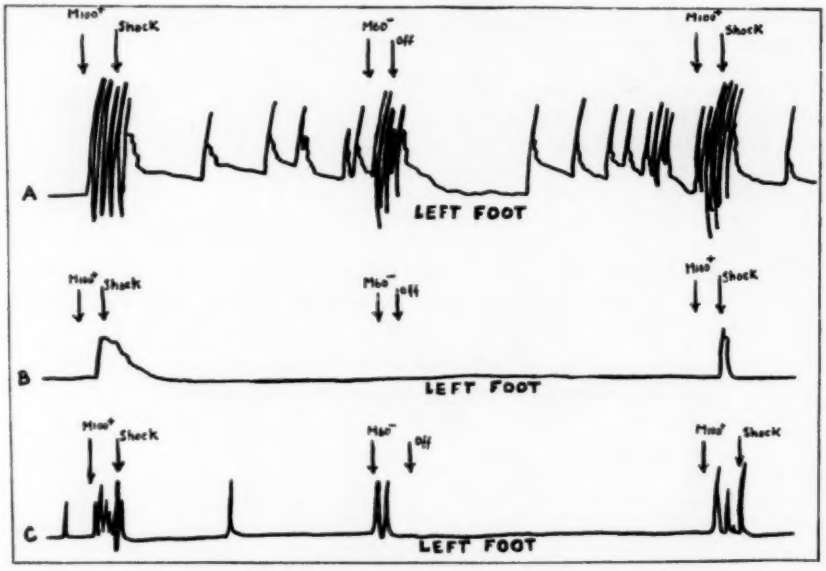


Fig. 7.—Effect of metrazol convulsions on the conditioned responses of a pathologically hyperactive dog.

A represents the preconvulsive period (hyperactivity, poor differentiation); *B* is a record on convulsion days, with improved differentiation due to decrease of activity, and *C*, a record after nine convulsions on an interval day (intermediate in activity between *A* and *B*).

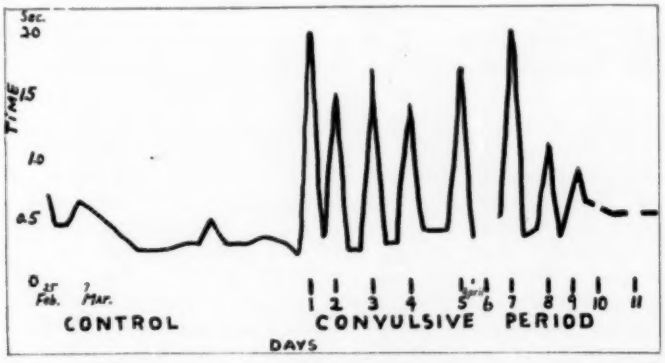


Fig. 8 (Laval).—Latent period of a conditioned defense reaction. The longer latent period after convulsions denotes slower reactions.

COMMENT

Metrazol convulsions in the dog, therefore, appear to alter, temporarily, the mentally integrated symbolic functions, represented by the conditioned responses. The alterations in the autonomic conditioned responses differ from those seen in the motor conditioned responses. The salivary conditioned reflex, mediated by a glandular–smooth muscle–parasympathetic neural mechanism, shows, after a

course of metrazol convulsions, first, a loss of differentiation between the food-reenforced and the non-food-reenforced stimulus, followed shortly by disappearance of the conditioned salivary responses in general. Coincidental with these changes there is a strong emergence of behavior responses mediated by lower levels of integrative activity, seen in the prolonged time of the orienting response and in the phenomenon of delayed excitation (the after-secretion [figs. 1, 2 and 3]). Wolff and Gantt [1] saw that in animals recovering from the effects of narcotic drugs the orienting reflex (a subcortical, unconditioned reflex) appeared while the animal was still lethargic, and before any of the conditioned responses had reappeared. Its continued presence is often evidence of suppression of higher nervous function.

On the defense reactions, mediated by striated, voluntary musculature, the motor nerves and the pyramidal pathways, the effect of the convulsions was somewhat different, depending chiefly on the type of animal. In the excitable dog there appeared to be an actual increase in the efficiency of the performance. This, however, was more apparent than real. The animal, as already stated, did give evidence of having made a differentiation between a positive stimulus of 100 a minute (M 100 +) and a negative stimulus of 60 a minute (M 60 —). He was unable, however, to inhibit completely the response to the negative stimulus. The convulsions appear to have increased the economy of this animal's performance by increasing the inhibitory tendency. It can be seen from figure 6 that this tendency also affected the reaction to the positive metronome stimulus. The dog reacted only from 70 to 90, rather than 100, per cent of the time, as he had in the control period. Further evidence of the decrease in general excitability is seen in the foot-lifting record and the prolongation of the latent period of the response. Furthermore, Connie also demonstrated this tendency. This animal, a stable dog, which learned to differentiate almost perfectly between the negative stimulus of 100 a minute (M 100 —) and the positive stimulus of 92 a minute (M 92 +) before the course of metrazol convulsions was started, showed a long latent period in the conditioned response, a single lift of the paw to the positive metronome stimulus and a minimum of random movements (fig. 5). In this animal the metrazol treatment produced the following behavior: In the post-convulsive period there was no reaction to either metronome beat. The dog merely stood still and received the shock on alternate stimuli. On the interval day there was progressive deterioration of his differentiating ability. In this stable dog, therefore, the increase of the inhibitory tendency caused a decrease in the efficiency and economy of his performance—the reverse of the effect in Laval.

The difficulty of evaluating the clinical improvement from shock therapy has been pointed out by Cobb [2] and many others, who have questioned whether the results with this method are much better than those with other forms of shock or with the careful attention of a personal physician, such as the patient would get in a private hospital. Considerable evidence exists for damage to the brain tissue as a result of the convulsion. Cobb [2c] stated:

> One suspects that the treatment is merely palliative and carried out at the risk of permanent damage to the brain . . . The basal ganglia are the chief seat of the hemorrhagic lesions, although they often occur in the cerebral cortex.

---

1. Wolff, H. G., and Gantt, W. H.: Caffeine Sodiobenzoate, Sodium Iso-Amylethyl Barbiturate, Sodium Bromide and Chloral Hydrate: Effect on Highest Integrative Functions, Arch. Neurol. & Psychiat. **33**:1030 (May) 1935.

2. Cobb, S.: (*a*) Review of Neuropsychiatry for 1938, Arch. Int. Med. **62**:883-899 (Nov.) 1938; (*b*) Review of Neuropsychiatry for 1941, ibid. **68**:1232-1245 (Dec.) 1941; (*c*) Shock Therapy, New England J. Med. **217**:195-196 (July 29) 1937.

Besides the histologic evidence, electroencephalographic records point to damage (Grinker, Levy and Serota [3]).

There is evidence that all the convulsant drugs produce a similar type of nervous impairment. Cobb expressed the belief that any convulsion results in damage to the brain.

In addition to the anatomic damage of brain tissue, our results point to a notable impairment of function—evident in the lessening of the intensity of the excitatory reactions, the retardation of the response and the long period (months) necessary for recovery.

Gellhorn and associates [4] have shown in rats that extinguished conditioned reflexes become spontaneously restored after convulsive shocks. On the face of it, their experimental results are in contradiction to ours, which point to a decrease of excitation. On careful analysis, however, the results are identical with ours: The restoration of an excitatory conditioned reflex is merely the conversion of an inhibitory response (the extinguished conditioned reflex) into an excitatory one, which is in reality what we saw in our dogs in the loss of differentiation, in which the inhibitory conditioned reflex becomes excitatory. Gellhorn worked with the inhibition of extinction while we used the inhibition of differentiation, but the effects were identical. The clearcut results in animals as different as the rat and the dog are striking.

On the other hand, Stainbrook and Löwenbach,[5] working with the rat, concluded that "the performance of the animal in so far as the retention of a recently acquired habit is concerned did not seem to be affected by a long series of convulsions." In comparing the results of these authors with Gellhorn's and ours, it is necessary to consider the degree of discrimination measured and the intensity of the motivation (unconditioned stimulus). With a method involving a crude form of differentiation or very strong motivation, the impairment might not be detected as readily as it would be by a finer differentiation or by not so strong a reenforcement of the unconditioned stimulus (motivation). For this reason, the results of Stainbrook and Löwenbach are not necessarily in conflict with those of Gellhorn and his associates and our own.

Two further considerations deserve emphasis: first, the normal or pathologic status of the specimen, and, second, the importance of the individual type. Thus, with our extremely excitable, restless dog the apparent results were the reverse of those we obtained with the normal, stable animals.[6] The improvement in the excitatory animal versus the impairment in the stable dog could be partly explained by the lessening effect of the convulsion on the excitatory process, together with the fact that the excitatory dog had never acquired efficient inhibitory reactions. If the lack of differentiation, i. e., the similarity of the responses to excitatory and to inhibitory stimuli, in the excitable dog, Laval is considered as representing two excitatory conditioned reflexes, instead of ex... ...on and inhibition, then the results are perfectly in accord with what we saw in the normal stable dogs. There is more justification for one's considering both responses excitatory than otherwise, for Laval never differentiated between the two stimuli.

---

3. Levy, N. A.; Serota, H. M., and Grinker, R. R.: Electroencephalographic and Clinical Studies Following Convulsive Shock Therapy of Affective Disorders, Arch. Neurol. & Psychiat. **46**:542-543 (Sept.) 1941.

4. Gellhorn, E.; Kessler, M., and Minatoya, H.: Influence of Metrazol, Insulin Hypoglycemia, and Electrically Induced Convulsions on Reestablishment of Inhibited Conditioned Reflexes, Proc. Soc. Exper. Biol. & Med. **50**:260-262 (June) 1942.

5. Stainbrook, E. J., and Löwenbach, H.: The Reorientation and Maze Behavior of the Rat After Noise-Fright and Electroshock Convulsions, J. Comp. Psychol. **34**:293 (Dec.) 1942.

6. Dr. C. F. Jacobsen also found decreased activity in monkeys after metrazol convulsions (cited by Rioch [Tr. Am. Neurol. A. **68**:44, 1942]).

The reversal of effect, i. e., the conversion of inhibition into excitation, is what Pavlov [7] noted in many states involving what he termed overstrain, or weakening, of the cortical cells, and he described it as the ultraparadoxic phase. It occurs as the result of two extremely strong conditioned stimuli acting on the animal, as well as of normal stimuli acting on a weak dog. These facts fit well with the known action of the convulsive drugs, viz., impairment of the cortical cells.

The return of more or less normal function after six or seven months is not incompatible with the irreparable destruction of nerve cells, for the brain has been shown to have compensatory function. Thus after extirpation of parts of the cortex, there is an immediate and enormous loss of conditioned reflex function, but a gradual restoration of at least part of the lost function in both dogs and patients.

Much has been written of the possible effect of fear in shock therapy. It is interesting to point out that 2 of our dogs became conditioned to the shock stimulus; i. e., they made twitching movements with shivering, resembling petit mal, when they were brought near the experimental room after they had had three or four convulsions. After so few as twelve convulsions, the appearance of a convulsion in response to a conditioned reflex might throw some light on certain forms of epileptic seizures.

Function after shock therapy in the patient has not been satisfactorily studied. However, Gantt and Muncie [8] observed serious amnesia in several patients as long as a year after the metrazol convulsions. These patients, like the Korsakovs, were completely unable to form new conditioned reflexes. The dogs we used were tested chiefly for retention of old conditioned reflexes, rather than for ability to form new ones. As the latter function is a more delicate one, it is probable that this suffered even more than the retention.

The histologic evidence of damage and the prolonged period of loss of function would not in themselves vitiate any beneficial effects of the shock therapy, but the double evidence of impairment (anatomic and functional) makes necessary a most careful evaluation of the probability of therapeutic benefit before such serious damage is inflicted on the brain tissues.

SUMMARY

The course of twelve metrazol convulsions gave varying results, depending on the type of animal. The effect was much more pronounced on the higher, conditioned reflex function than on the lower, unconditioned activity. In general, there was impairment of cerebral function in the direction of (1) decrease of excitatory conditioned reflexes; (2) loss of inhibition, resulting in lack of differentiation between positive and negative stimuli; (3) lengthening of the latent period; (4) predominance of the activity of some lower centers at the expense of the higher ones, and (5) a long period of recovery. The impairment of function lasted from one to six months, even in the stable dogs. In 1 extremely excitatory dog, in which inhibition was poor, the lessening of the excitatory conditioned reflexes resulted in apparent improvement in the behavior, rather than in impairment.

Johns Hopkins Hospital.

7. Pavlov, I. P.: Lectures on Conditioned Reflexes: Conditioned Reflexes and Psychiatry, translated and edited by W. Horsley Gantt, New York, International Publishers, 1941, vol. 2.

8. Gantt, W. H., and Muncie, W.: Analysis of the Mental Defect in Chronic Korsakov's Psychosis by Means of the Conditioned Reflex Method, Bull. Johns Hopkins Hosp. **70**:467-487 (June) 1942.

CLINICAL AND ELECTROENCEPHALOGRAPHIC STUDIES ON CRIMINAL PSYCHOPATHS

DANIEL SILVERMAN, M.D.
Assistant Surgeon (Reserve), United States Public Health Service
SPRINGFIELD, MO.

The concept of the psychopathic personality, long a controversial subject in psychiatry, has been greatly clarified in the recent literature. However, there are still disagreement about the delineation of the nosologic group and wide divergence of opinion about the essential psychopathology of the disorder. My object in this investigation was to study the etiologic factors, both organic and psychogenic, which were operative in a selected group of "typical" psychopaths, after I had arrived at a working definition of psychopathy which would be clinically distinct and homogeneous (though the disturbances of the various patients might vary greatly). As far as could be determined, this is the first application of electroencephalography to the study of adult psychopaths. In selection of the subjects no attention was paid to possible psychodynamic factors unless the resulting clinical state suggested neurosis or psychosis, in which case the patient was excluded from the investigation. Likewise, persons who had obvious evidence of organic dysfunction of the brain, such as focal neurologic signs, depressed fracture of the skull or epileptic seizures, were excluded. The cases of the patients retained for study were reviewed by the neuropsychiatric staff, and unanimity of opinion on the diagnosis of psychopathic personality was required.

The patients in this study were all residents of the psychopathic unit at the Medical Center for Federal Prisoners, a general and psychiatric hospital which receives as patients male inmates from all federal prisons and reformatories. The historical data were obtained from interviews, examinations and observations at the Medical Center, from questionnaires sent to friends and relatives, from home social service investigations and from institutional records. In addition to undergoing neurologic examinations, with negative results, the patients were reexamined and requestioned for suggestions of neurologic disease. Finally, electroencephalograms were taken on each patient.

DIAGNOSTIC CRITERIA

The criteria used in this study were in essential agreement with those of Cleckley,[1] Maughs,[2] and Caldwell[3] for the noncriminal psychopathic personality, and no fundamental difference was found between the criminal and the noncriminal psychopath. In the clinicodescriptive definition which follows, an attempt was made to outline the predominant traits. Not all the psychopathic personalities in this study were as extreme as the condition pictured.

In the absence of psychotic symptoms or a definite neurosis, noteworthy abnormalities in behavior are the signposts for recognition of the psychopath. A

From the United States Public Health Service, Medical Center for Federal Prisoners.

1. Cleckley, H.: The Mask of Sanity, St. Louis, C. V. Mosby Company, 1941.

2. Maughs, S.: A Concept of Psychopathy and Psychopathic Personality: Its Evolutional Historical Development, J. Crim. Psychopath. **2**:329-356 and 465-499, 1941; **3**:495-516, 1942.

3. Caldwell, J. M., Jr.: The Constitutional Psychopathic State, J. Crim. Psychopath. **3**:171-179, 1941.

longitudinal review of his life reveals behavior which is reckless, impulsive, unrepressed and often bizarre and pointless. In relation to society he is unproductive, parasitic and antisocial. In relation to his fellow men he is insincere, untrustworthy, irresponsible, sometimes overbearing and demanding and often outright hostile. Affect is cold, humorless and lacking in qualities of genuine warmth, gratitude and remorse. Close interpersonal relationships are never developed, and the psychopath remains extremely narcissistic and selfish. Sexual relationships are promiscuous, inadequate (deficient) or perverse and are always lacking in real feeling tone for the object. Efforts are unsustained; interests are fragmentary, and there is lack of any consistent life plan. Although sometimes there is an appearance of brilliance, thinking is superficial and at a plane far below that anticipated from the psychometric test level. If the patient recognizes his difficulties at all, projection thinking is the commonest method of rationalizing them.

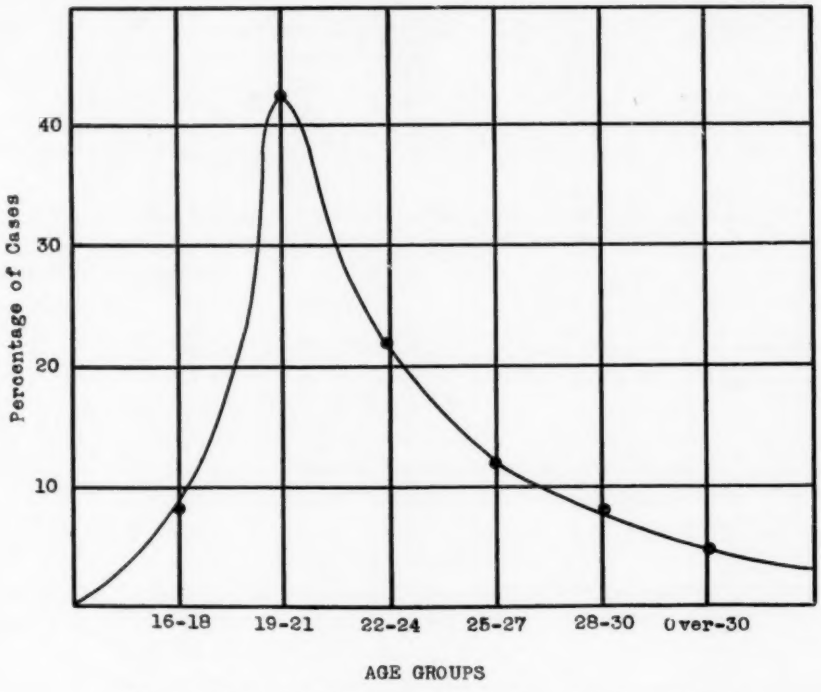


Fig. 1.—Age distribution curve for 75 criminal psychopaths.

There is often total disregard for the truth. Insight is usually absent, although one is sometimes confronted with superficial verbal insight. Anxiety is rarely manifest, and then only in response to situational difficulty (e. g., incarceration) which the psychopath has brought on himself. Tolerance to frustration or tedium is poor, and this trait may result in unprofitable, impulsive violent acts, temper tantrums or evanescent psychotic-like behavior. Despite their unsavory and antisocial qualities, on cross sectional review many psychopaths appear superficially intact, pleasant and likable. It is often only by long contact that the underlying chaotic and sick personality is revealed.

STATISTICAL CHARACTERISTICS OF THE GROUP

The ages of the 75 patients varied from 16 to 43 years; the age distribution curve is shown in figure 1. The highest incidence (32, or 43 per cent) was

between the ages of 19 and 22 years; this corresponded to the age incidence of most frequent criminality.[4] The mental ages of 70 of the subjects were determined by the Army alpha and/or the Stanford-Binet test. The distribution was similar to that of the general population averages, except that there were more persons with normal, but fewer with dull normal and bright normal, intelligence (table 1). Although the men were regarded as a danger to society, 65, or 87 per cent, of their offenses which resulted in incarceration were nonviolent crimes of acquisi-

TABLE 1.—*Distribution of Intelligence Ratings for Seventy-Five Criminal Psychopaths*

Intelligence	Mental Age, Yr.	Intelligence Quotient	Number of Patients	Percentage in This Group	Percentage in General Population
Feebleminded	0- 7	0-50	0	0.0	1
	7-10	51-70	2	2.9	
Borderline	10-11	71-75	1	1.4	5
Dull normal	11-13	76-87	9	12.9	24
Normal	13-17	88-113	40	57.1	40
Bright normal	17-18	114-120	9	12.9	20
Superior	18-21	121-140	8	11.4	8
Very superior	Over 21	Over 140	1	1.4	2
Unknown	.....	.....	5		
			75		

TABLE 2.—*Offenses Committed by Seventy-Five Criminal Psychopaths*

	Number	Percentage
Crimes of acquisitiveness, without violence	65	86.7
Thefts	58	77.3
Automobile	43	
Mail	8	
Interstate	3	
Other	4	
Swindles	5	6.7
Forgery	2	
Impersonation	1	
Mail fraud	1	
Counterfeiting	1	
Illicit business	2	2.7
Liquor	1	
White slave	1	
Crimes involving violence, actual or implied	9	12.0
For money	4	5.3
Bank robbery	1	
Other robbery	1	
Extortion	2	
Not for money	2	2.7
Assault	2	
Crimes against sex morals	3	4.0
Sodomy	2	
Rape	1	
Miscellaneous (immigration violation)	1	1.3

tiveness (table 2). Automobile theft (chiefly a violation of the Dyer Act) was by far the most frequent offense, 57 per cent, but in very few cases was this act undertaken for pecuniary gain. Characteristically, the offender stated that he committed the theft impulsively—"to take a joy ride" or to get from one state to another, since "no one would give me a lift"; sometimes a car was stolen for no observable purpose and wrecked while he was drinking, or sometimes it was stolen simply because he could not resist an empty car with the keys left in it. Sometimes, however, the car was stolen as a means of escape from the state or

4. Statistical Abstract of the United States Census, United States Department of Commerce, Bureau of Census, 1940, p. 76.

local police, or as an aid to a rampage of burglaries and robberies. The sentences received were longer than the average federal sentence, probably because of the criminal psychopath's unsavory previous record; 30 offenders had sentences of between one and three years; 28, sentences of over three and up to five years, and the remainder had longer sentences. Nine of the psychopaths with the longer terms incurred additional time for escapes from federal custody. One third had long previous criminal records, with two or more penal experiences; another third had served one sentence in a penitentiary or reformatory; 16 men had served only jail sentences, and 9 had no prior criminal record. The high degree of recidivism (88 per cent) is noteworthy.

The majority of the psychopathic criminals, 46 (61 per cent), came from the large and small urban communities. The regional distribution was nation wide; all were native born except an English-born American, a Canadian and a Mexican. Two patients were Negroes. The economic status of the homes in which the patients were reared was submarginal for 40 per cent, marginal for 39 per cent and comfortable for 16 per cent; it was not stated for 15 per cent. Forty-seven (63 per cent) had a "broken home" or had suffered the loss or absence of one or both parents before he reached the age of 15 years. Such mentally unhygienic factors as parental cruelty, immorality, criminality, insanity or pronounced ambivalance, rejection or overindulgence (rare—5 patients) characterized the histories of 48 patients (64 per cent). Only 15 (20 per cent) of the offenders had a presumably nonpathologic background. Only 13 (17 per cent) of the subjects, on the other hand, had criminal siblings, and mental disease or defects in siblings were uncommon (4 or 5 per cent).

Severe abnormalities of behavior were clearly evident at an early age in 49 (65 per cent) of the patients, and milder disorders, in 16 (21 per cent). Eight (11 per cent) first manifested psychopathic traits in adolescence, and only 2 had no definite history of psychopathy until adult life. The educational record of the group was poor; 35 (47 per cent) did not complete grade school, and only 6 (8 per cent) finished high school. None advanced beyond the secondary schools. The occupational adjustments were almost uniformly poor; only 1 had a good work record, and 10 were considered fair, albeit unstable, workers. Forty-one (55 per cent) showed marked residential instability (transiency). A history of alcoholism was obtained for 32 subjects, but in only 4 was the symptom prominent. Two patients were intermittent addicts to the use of narcotics. Twenty-five (33 per cent) had a sexual history of marked promiscuity, and 23 (31 per cent), of homosexual indulgences (8 of these had pronounced homosexual tendencies); only 2 of the 15 men who attempted marriage had any successful relationship. In the outside world few showed conventional symptoms of mental disturbance, but on conviction or incarceration 13 were hospitalized for "psychosis" and 5 for "severe neuroticism." As an indication of the psychopath's inability to get along under a repressive environment, 68 of the 75 patients repeatedly violated institutional rules and had to be kept under strict supervision in order to prevent them from violating rules or disturbing the morale of other inmates. Seventeen patients exhibited maladjustments by acts of the less aggressive, or minor, type, such as conniving, late sleeping, refusal to work and insolence, but for 51 subjects the maladjustment was of a more serious nature, involving escape, assault, fighting, insubordination, creation of disturbance and destruction of property, with resultant loss of time for good behavior for 33 men. Twenty-six of the men had viciously assaulted members of the personnel or other inmates. It is obvious from this superficial analysis that the case material included examples of the most extreme and dangerous criminal psychopathic types.

ELECTROENCEPHALOGRAPHIC CHARACTERISTICS

The apparatus used was a five channel, push-pull, resistance-capacity–coupled, pen-recording electroencephalograph. Electrode placements, made according to the technic of Jasper,[5] were employed to cover the prefrontal, frontal, central or motor, parietal and occipital areas of the scalp, both for bipolar and for "monopolar" (common ear) leads. Twenty-two electrodes were used in the routine examination. Two to three minute tracings were made on each side with bipolar and then with monopolar leads. This was followed by simultaneous recordings from homol-

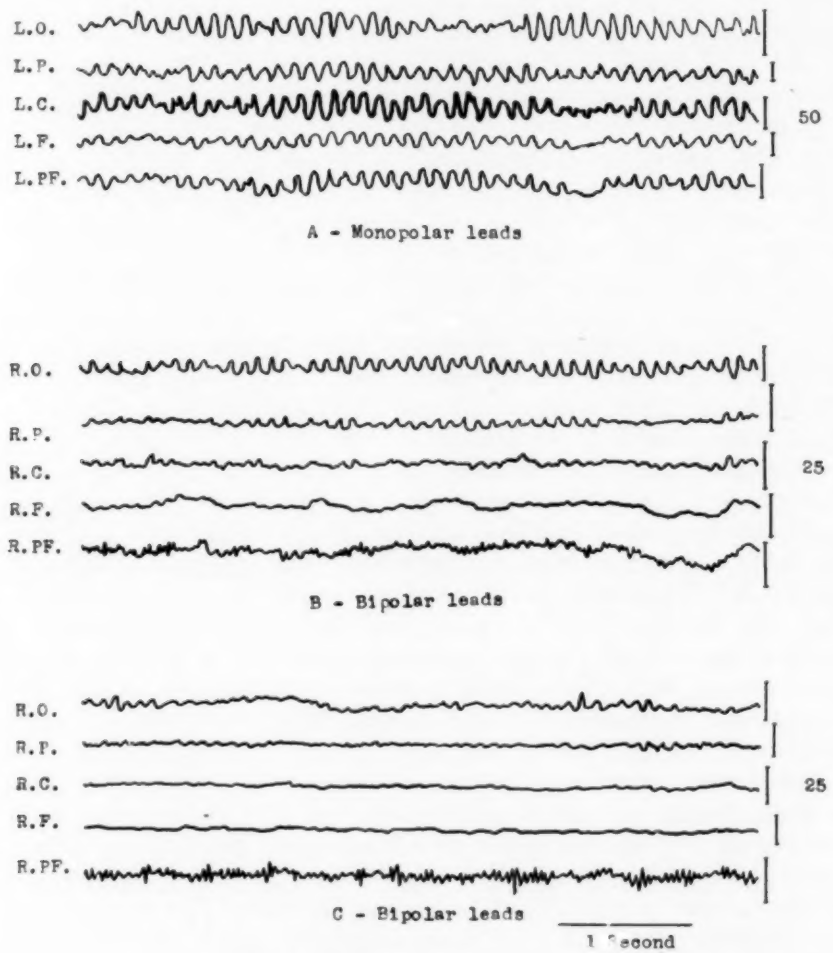


Fig. 2.—Normal rhythms.

In this figure, and in figures 3 and 4, calibrations are in microvolts, and time is measured in seconds.

ogous areas in the two hemispheres and by the taking of an anterior-posterior tracing (prefrontal to occiput, central to occiput and prefrontal to central). At the end of this procedure the patient hyperventilated for two minutes, and a tracing was made during the last minute of hyperventilation and for at least a minute thereafter. The minimum time for a total record was eighteen minutes.

5. Jasper, H. H., in Penfield, W., and Erickson, T.: Epilepsy and Cerebral Localization, Springfield, Ill., Charles C Thomas, Publisher, 1941, pp. 383-387.

If pathologic waves were observed, a technic [6] for localization by phase reversals was employed. With selected patients having prominent pathologic waves an attempt was made to determine the possible origin of the abnormal activity from the basal, or hypothalamic lead.[7] A total of 96 tracings were made on 75 psychopathic criminals.

The records were divided into three main types: the normal, the borderline abnormal and the abnormal. The criterion for classification of abnormal records was the presence of a definite delta rhythm of a frequency below 7.5 per second

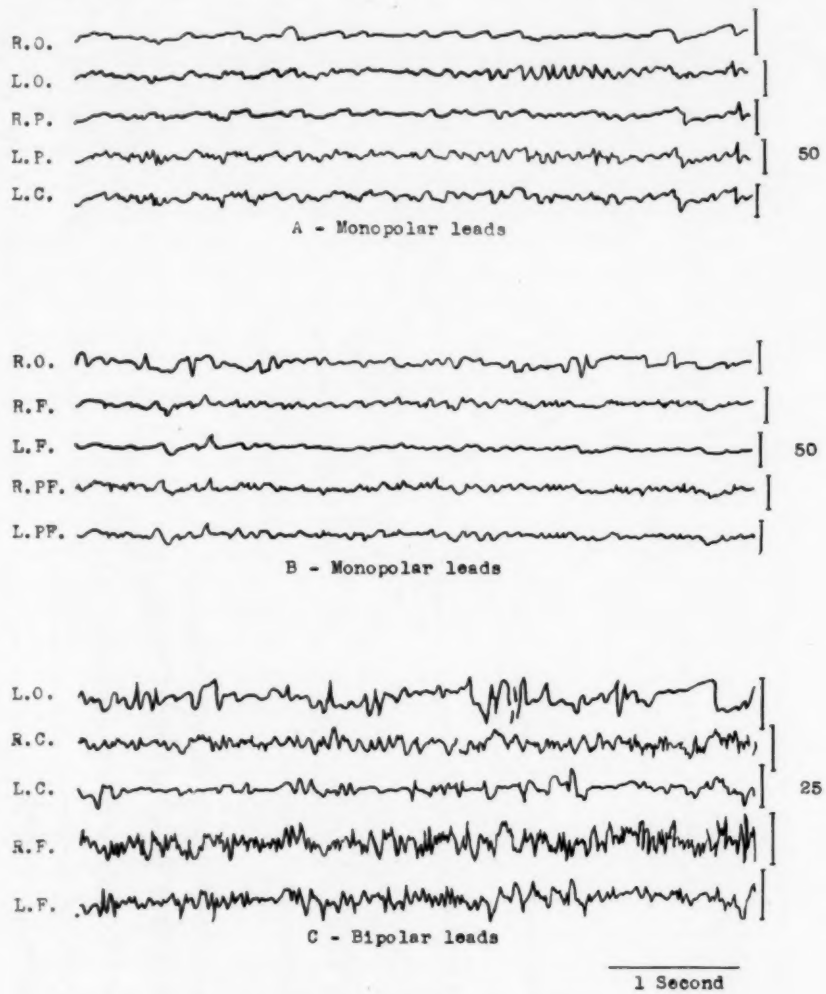


Fig. 3.—Borderline abnormal rhythms.

and/or the occurrence of definite high voltage spike activity in roughly more than 10 per cent of the record. It was not thought advantageous to classify these records further into the very abnormal and the abnormal, as was done by Finley and Campbell.[8] The borderline type was characterized by arrhythmia. Random

6. Walter, W. G.: The Location of Cerebral Tumors by Electroencephalography, Lancet **2**:305-308, 1936.

7. Grinker, R. R.: A Method for Studying and Influencing Corticohypothalamic Relations, Science **87**:73-74, 1938.

8. Finley, K. H., and Campbell, C. M.: Electroencephalography in Schizophrenia, Am. J. Psychiat. **98**:374-381, 1941.

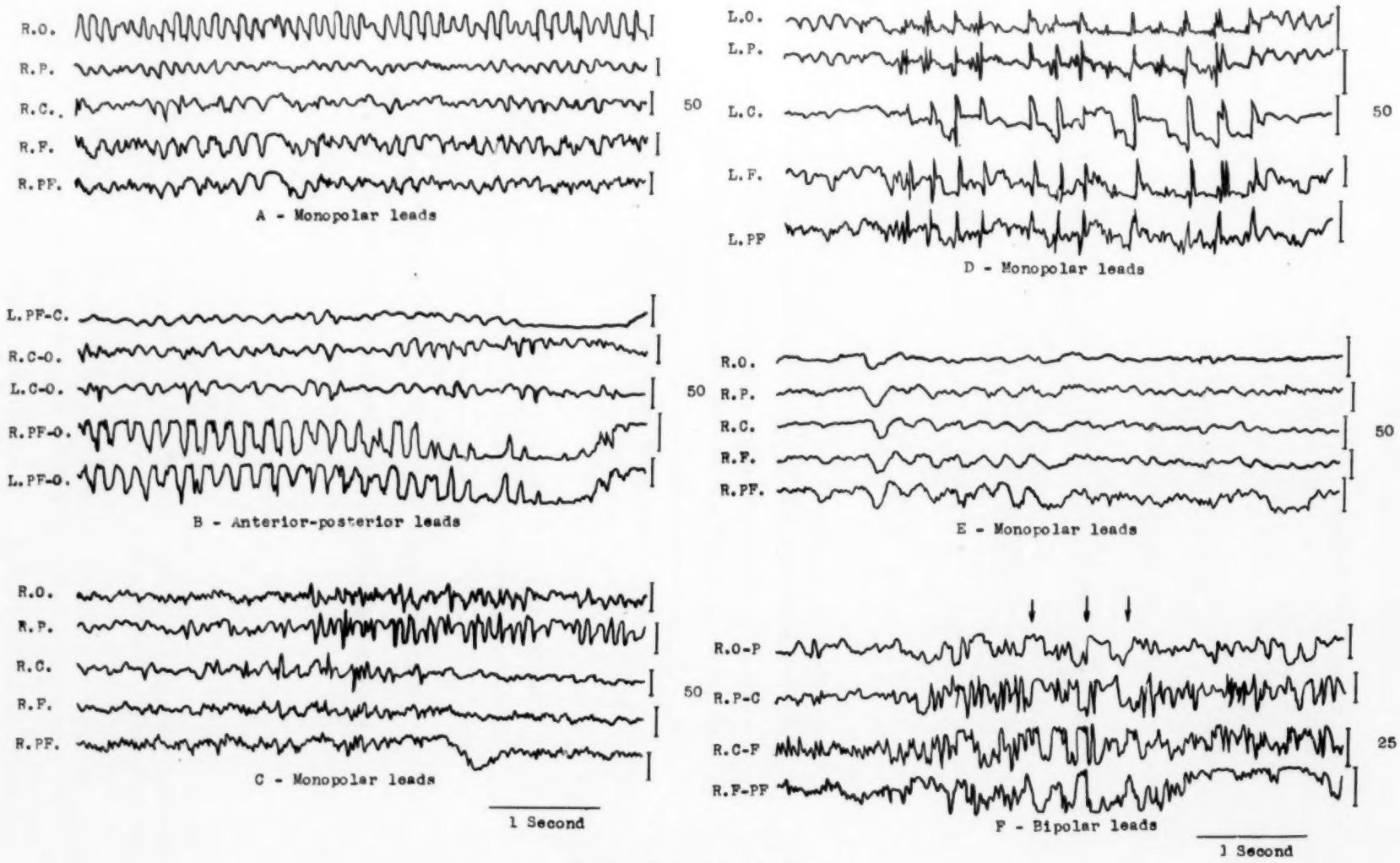


Fig. 4.—Abnormal rhythms.

slow waves and spikes might appear in these records but did not form a definite rhythm. Asynchronism between the hemispheres might be present. Records in which abnormal rhythms appeared only on hyperventilation were also included in the borderline group. The normal records possessed none of the features of the other two in any prominence (in less than 10 per cent of the record). In the normal tracing there appeared a regular series of smooth alpha waves (8 to 12 per second), more continuously in the parieto-occipital area, and beta waves (18 to 32 per second), chiefly in the anterior regions of the head.

Of the 75 psychopaths, only 15, or 20 per cent, had tracings classifiable as normal. Nine of these 15 patients had some, but an insignificant number of, random slow waves and/or spikes. Nine of the subjects had records of the dominant and subdominant alpha type (fig. 2 *A*), 4 of the mixed alpha type (fig. 2 *B*) and 2 of the poor alpha type (fig. 2 *C*). Although the group was too small for analysis, the patterns followed the distribution of presumably normal persons.[9] Twenty, or 26.6 per cent, had tracings which fell in the borderline classification. Nine of these had pronounced arrhythmia anteriorly (i. e., in the prefrontal and frontal regions) and 1 posteriorly (in the occiput), and 10 had diffuse arrhythmia (fig. 3 *B*). In 8 subjects there was a tendency for a 6 per second delta rhythm to appear anteriorly, and in 6, for spikes to occur posteriorly, but these abnormalities constituted less than 10 per cent of the record. Four of the group manifested asynchronism between the hemispheres (fig. 3 *A*), and 4, striking abnormalities on hyperventilation (fig. 3 *C*).

The bulk of the tracings, 40, or 53.4 per cent, were definitely abnormal. In half of them the striking abnormality was the presence of a definite 6 per second rhythm originating diffusely from the frontal and the prefrontal area of the brain (fig. 4 *A* and *B*). Slow rhythms of various frequencies and of diffuse origin were present in 8 tracings (fig. 4 *E*); diffuse (but more posterior) spike activity predominated in 8 (fig. 4 *C*); slow wave and spike activity (fig. 4 *D*), suggestive of petit mal,[10] was present in 2, and localized pathologic waves (fig. 4 *F*), with insufficient clinical evidence to warrant a neurologic diagnosis, appeared in 2. None of the abnormal waves examined with the basal lead technic were localized in the hypothalmic area.

Fig. 4.—Abnormal rhythms.

THE CASE FOR AN ORGANIC CAUSE

In the early literature the belief predominated that the essential pathology of the psychopathic personality, then called the moral insane, or moral imbecile, was an inborn structural defect. Maudsley,[11] in 1896, noted that the psychopathic behavior was often a premonitory syndrome of organic disease of the brain, such as dementia paralytica or senile dementia, and concluded that "many cases of moral insanity will be found to be connected with more or less congenital moral defects." Later investigators observed that many psychopaths were congenitally defective in general, and this idea persisted in the term "psychic constitutional inferiority" of Adolf Meyer.[12] Healy's[13] early work emphasized this aspect, and Sadler[14]

9. Davis, H., and Davis, P. A.: Action Potentials of the Brain in Normal Persons and in Normal States of Cerebral Activity, Arch. Neurol. & Psychiat. **36**:1214-1224 (Dec.) 1936; The Electrical Activity of the Brain: Its Relation to Physiological States and to States of Impaired Consciousness, A. Research Nerv. & Ment. Dis., Proc. (1938) **19**:50-80, 1939.

10. Gibbs, F. A.; Lennox, W. G., and Gibbs, E. L.: Localizable Features of the Electrical Activity of the Brain in Petit Mal Epilepsy, Am. J. Physiol. **116**:61, 1936.

11. Maudsley, H.: Responsibility in Mental Disease, London, King & Co., 1896.

12. Meyer, A.: Constitutional Abnormality, in Oberndorf, C. P.: Discussion, State Hospital Bulletin, March 1910; cited by Cleckley,[1] p. 176.

(Footnotes continued on next page)

expressed the opinion that psychopaths were "neurologically disinherited from birth." Later many psychiatrists [15] observed psychopathic-like states after birth injuries, trauma to the brain in childhood and encephalitides. Other, more sanguine writers sought precise localization of the defect; Browning,[16] basing his ideas on the results of frontal lobe surgery, expressed the belief that the "moral center" was localized in the right frontal lobe; Kahn [17] stated that the psychopathic personality might be a brain stem syndrome ("organic drivenness"); Ingham [18] claimed it was due to a defect of the diencephalon, and Chornyak,[19] to a defect in the "most recently acquired areas of the cerebrum," the angular and supramarginal gyri. He concluded that these areas were selectively vulnerable to damage by anoxia, early trauma, or perhaps congenital anomaly, because the cells at birth and in early youth were still in an undifferentiated, embryonic stage of growth. Rosanoff and associates,[20] in a study of dizygotic and monozygotic twins, found considerable statistical evidence for cerebral birth trauma as an etiologic factor in behavior difficulties in childhood; by virtue of the greater vulnerability to trauma of cerebral tissues in males,[21] they accounted for the preponderance of behavior problems and delinquencies in male children.

In this study, the case histories of the 75 psychopaths were carefully rechecked for evidence of (*a*) a family history of epilepsy, (*b*) congenital defects, (*c*) birth trauma or anoxia and (*d*) significant trauma to the brain, anoxia or infectious encephalopathy occurring before the age of 5 years. In 15 patients a cerebral pathologic process was thought possible, and in an additional 5 it was believed probable. Six other patients possibly suffered significant cerebral damage after the age of 5 years and 1 probably did. The data from the histories are summarized in table 3. Neurologic reexamination of the 75 patients revealed definite signs of nonspecific disease of the central nervous system in 3 patients and suggestive signs in 18 patients. The neurologic findings are summarized in table 4. A history of prolonged enuresis, though it occurred frequently (33 per cent), was not included as a positive neurologic sign because of the psychogenic aspects of this symptom. It should be noted that the signs were almost uniformly confined

13. Healy, W.: The Individual Delinquent, Boston, Little, Brown & Company, 1920, pp. 188-190.

14. Sadler, W. S.: Theory and Practice of Psychiatry, St. Louis, C. V. Mosby Company, 1936, p. 775.

15. Ebaugh, F. G.: Neuropsychiatric Sequelae of Acute Epidemic Encephalitis in Children, Am. J. Dis. Child. **25**:89-97 (Feb.) 1923. Bender, L.: Anatomic-Pathologic Data on Personality Function, Am. J. Psychiat. **92**:328-351, 1935. Karnosh, L. J.: Constitutional Psychopathic Personality: Their Recognition, Diagnosis and Management, M. Clin. North America **19**:1935-1937, 1936. Blau, A.: Mental Changes Following Head Trauma in Children, Arch. Neurol. & Psychiat. **35**:723-769 (April) 1936. Other psychiatrists made similar observations.

16. Browning, W.: The Moral Center in the Brain: Its Localization and Significance, M. Rec. **99**:1043-1048, 1921.

17. Kahn, E., and Cohen, L. H.: Organic Drivenness: A Brain-Stem Syndrome and an Experience, New England J. Med. **210**:748-756, 1934.

18. Ingham, S. D.: Some Neurologic Aspects of Psychiatry, J. A. M. A. **111**:665-668 (Aug. 20) 1938.

19. Chornyak, J.: Some Remarks on the Diagnosis of Psychopathic Delinquents, Am. J. Psychiat. **97**:1326-1340, 1941; cited by Healy, W.: Personality in Formation and Action, New York, W. W. Norton & Company, Inc., 1938, pp. 109-110.

20. Rosanoff, A. J.; Hardy, L. M., and Plesset, I. R.: The Etiology of Child Behavior Difficulties, Juvenile Delinquency and Adult Criminality, with Special Reference to Their Occurrence in Twins, State of California Department of Institutions Psychiatric Monographs, Sacramento, California State Printing Office, 1941, no. 1.

21. Rosanoff, A. J.; Hardy, L. M., and Plesset, I. R.: The Etiology of Mental Deficiency, with Special Reference to Its Occurrence in Twins, Psychological Monographs, Princeton, N. J., Psychological Review Company, 1937, vol. 48, no. 4.

to the motor and extrapyramidal systems. The total number of patients with neurologic signs and/or histories suggestive of a cerebral lesion was 39 (53 per cent). It must be emphasized that those patients were considered typical psychopaths, and not persons with disturbances resulting from an organic lesion in the brain. Correlation of the clinical evidence of cerebral lesions with the electroencephalograms is given in table 5; the occurrence of abnormal brain rhythms was no more frequent in the patients with evidence of "organic" disease than in patients without such evidence.

TABLE 3.—*Evidence of Cerebral Lesion in Histories of Twenty-Seven Criminal Psychopaths*

Family history of epilepsy	2
Congenital defects (minor)	3
Dystocia	9
Birth trauma	5
Head injury, under 5 years of age	3
Head injury, over 5 years of age	7
Severe infection during infancy	3
Severe malnutrition during infancy	1

TABLE 4.—*Neurologic Signs Observed in Twenty-One Criminal Psychopaths*

Inequality of deep tendon reflexes	7
Inequality of superficial abdominal reflexes	2
Questionable pathologic reflexes (Hoffmann, resistance, Babinski, etc.)	7
Pathologic reflexes	3
Irregularity and reflex inequality of pupils	2
Internal strabismus (congenital)	2
Unilateral weakness of face	4
Severe tremor	9
Incoordination (finger to nose test)	2
Impaired associated movements, unilateral	5
Dysarthria	5

TABLE 5.—*Correlation of Neurologic Signs and a History Suggestive of Cerebral Lesion with the Type of Electroencephalogram for Seventy-Five Criminal Psychopaths*

	Electroencephalogram		
	Normal	Borderline Abnormal	Abnormal
History and/or neurologic signs suggestive of cerebral lesion (39 cases)	8 (20%)	10 (26%)	21 (54%)
History suggestive of cerebral lesion (27 cases)	8	6	13
Neurologic signs suggestive of cerebral lesion (21 cases)	3	7	11

At the time of writing the most conclusive evidence for an organic (cerebral) basis for the psychopathic personality seems to be in the electroencephalogram. It is well known that structural alterations of the cerebral cortex are accompanied by abnormal brain rhythms, which apparently arise from malfunctioning adjacent "normal" brain tissue.[22] It is also known that certain diseases, such as idiopathic epilepsy, with which no cortical structural changes have been found to be associated, are accompanied by a high percentage of pathologic electroencephalograms. Presumably, abnormal brain waves indicate physicochemical dysfunction of the cortex, whether or not they are based on a microscopically detectable defect. The arrhythmias of the borderline group are also found in the organic disease of the

22. Pacella, B. L., and Barrera, S. E.: Electroencephalography: Its Applications in Neurology and Psychiatry, Psychiatric Quart. **15**:407-437, 1941.

brain, and it has been suggested that such tracings are similar to records from the undeveloped cortex of early childhood.[23] Finally, a normal electroencephalogram does not exclude cerebral dysfunction, for it is well known that a subcortical pathologic process usually does not have its representation in the cortical electroencephalogram. According to this study, 80 per cent of the patients had abnormal or borderline abnormal brain rhythms and were suffering from cerebral dysfunction, whether it was inherent, congenital or acquired in later life. If the 10 per cent with normal tracings, but with signs suggestive of organic disease of the brain, are added to this 80 per cent, an impressive total of 90 per cent of the psychopathic subjects studied had a significant detectable organic component to their illness.

These electroencephalographic alterations observed in psychopaths are corroborated by the results of previous studies on behavior problem children or the potential psychopaths. Jasper and his co-workers[24] first reported on the electroencephalograms of behavior problem children; in their group of 71 children (aged 2 to 16 years) they found that 73 per cent of the records were abnormal or borderline abnormal, and they described the most prominent abnormality as a 5 to 6 per second rhythm originating from the central and frontal regions of the head. Later investigators[25] confirmed these observations. The striking feature in the abnormal records of the psychopaths was, similarly, a prominent 6 per second rhythm originating in the anterior half of the brain. From these data it may be inferred that the psychopath possesses a brain which is malfunctioning, and which has been malfunctioning since early childhood.

THE CASE FOR A PYSCHOGENIC ORIGIN

With the advent of the dynamic schools of psychiatry it was inevitable that a new insight into the cause of the psychopathic personality would be gained. Partridge[26] noted that the psychopath made strong (oral) demands on environment, demands unchecked by a superego and accompanied by feelings of inferiority and insecurity. Psychopathic symptoms were merely infantile ways by which he might dominate situations. Alexander[27] emphasized the role of an unconscious sense of guilt, with resulting self punishment (antisocial, self-destructive behavior). Wittels[28] expressed the belief that the psychopath remains at a phallic character level, owing to disturbed parental relationships, and that

23. Brill, N. Q., and Seidemann, H.: The Electroencephalogram of Normal Children, Am. J. Psychiat. **98**:250-256, 1941.

24. Jasper, H. H.; Solomon, P., and Bradley, C.: Electroencephalographic Analyses of Behavior Problem Children, Am. J. Psychiat. **95**:641-658, 1938.

25. Strauss, H.; Rahm, W. E., and Barrera, S. E.: Studies in a Group of Children with Psychiatric Disorders: I. Electroencephalographic Studies, Psychosom. Med. **2**:34-42, 1940. Lindsley, D. S., and Cutts, K. K.: Electroencephalograms of "Constitutionally Inferior" and Behavior Problem Children, Arch. Neurol. & Psychiat. **44**:1199-1212 (Dec.) 1940. Brown, W. T., and Solomon, C. E.: Delinquency and the Electroencephalograph, Am. J. Psychiat. **98**: 499-503, 1942. Brill, N. Q.; Seidemann, H.; Montague, H., and Balser, B. H.: Electroencephalographic Studies in Delinquent Behavior Problem Children, ibid. **98**:494-498, 1942. Gallagher, J. R.; Gibbs, E. L., and Gibbs, F. A.: Electrical Activity of the Cortex and the Personality in Adolescent Boys, Psychosom. Med. **4**:134-140, 1942.

26. Partridge, G. E.: A Study of Fifty Cases of Psychopathic Personality, Am. J. Psychiat. **7**:593-973, 1928.

27. Alexander, F.: The Psychoanalysis of the Total Personality, New York, Nervous and Mental Disease Publishing Company, 1930.

28. Wittels, F.: The Criminal Psychopath in the Psychoanalytic System, Psychoanalyt. Rev. **24**:276-291, 1937.

psychopathic activities serve as desexualized sexual wishes. Menninger [29] stressed the overwhelming hate impulses of the psychopath, which he controls only by a continual pretense. Caldwell [30] expressed the opinion that the origin of psychopathy lies in the failure of the child to solve the Oedipus situation, and Cleckley,[31] that the psychopath has faulty psychobiologic reactions early in life which lead to persistence of infantile behavior patterns and failure to develop a superego. Bender,[32] from her studies on children, clearly formulated a dynamic approach:

> If the care [especially maternal] is interrupted at too early an age the pattern [of normal development] becomes shattered and the child's personality and superego become arrested at an infantile level, and he may develop into what is known as a psychopathic personality in adult life.

Recently Karpman [33] proposed that the condition of most psychopaths be labeled symptomatic and that in a small remaining group (the "utterly and completely selfish"), in whom no psychogenic factors could be found, the disorder should be called idiopathic.

A syndrome as various as the psychopathic personality is difficult to reduce to a common denominator. Authors in the past have described as many as sixteen clinical types. However, it has become increasingly important to determine underlying mechanisms rather than to distinguish clinical types. From the dynamic viewpoint two problems present themselves: (1) the dominant motivating forces of psychopathic behavior and (2) the ways in which these forces have developed.

In the case material of this study there were three dominant motivating forces: hostility (aggressiveness, hate), hedonism (selfishness, narcissism) and self destructiveness (inadequacy, feelings of inferiority, unconscious guilt). Occasionally a fourth dynamism—a great need for love and attention—was an important part of the picture (case 1). Only to the extent that persons were motivated primarily by excessive hostility, excessive hedonism or excessive self destructiveness did it seem logical to classify the patients under these three heads. Of the 75 psychopaths, 32 were considered hostile, 27 hedonistic and 16 inadequate. Cases illustrating these three types are reported later. Except for patients of the inadequate type, for whom a slightly greater number of normal electroencephalograms were found (33 per cent), there was no correlation between the electroencephalogram and the type of psychopathic personality. Likewise, the severity of the symptoms bore no relation to the degree of abnormality of the electroencephalogram.

The personalities of 35 patients lent themselves to psychodynamic speculation. Severe emotional deprivation early in childhood, similar to the primary affect hunger of Levy,[34] was most prominent in 26 per cent, and the reaction usually was the development of the hostile pattern of psychopathy (case 1). Thirty-one per cent had evidence of marked emotional insecurity during childhood. Here maladjusted, usually poverty-stricken parents, alcoholic fathers or inadequate mothers and broken homes did not afford the child the security necessary for maturation or the parental ego ideals necessary for development of the superego,

---

29. Menninger, K. A.: Recognizing and Renaming Psychopathic Personalities, Bull. Menninger Clin. **5**:150-156, 1941.

30. Caldwell, J., Jr., cited by Cleckley,[1] pp. 196-197.

31. Cleckley,[1] pp. 274-275.

32. Bender, L.: Emotional Problems in Children, in Proceedings of the Second Institute on the Exceptional Child of the Child Research Clinic, The Woods School, Langhorne, Pa., 1935, pp. 49-64; cited by Curran and Schilder.[36]

33. Karpman, B.: On the Need of Separating Psychopathy into Two Distinct Clinical Types: The Symptomatic and the Idiopathic, J. Crim. Psychopath. **3**:112-137, 1941.

34. Levy, D. M.: Primary Affect Hunger, Am. J. Psychiat. **94**:643-652, 1937.

and the child became fixed either in a hostile or in an inadequate behavior pattern. Thirty-one per cent gave indications of severe, unresolved Oedipus conflicts and manifested the hostile, the narcissistic or the inadequate behavior pattern (case 3). A few (11 per cent) showed oral fixation traceable to an extremely overindulgent parent (usually the mother), similar to the mechanism in chronic alcoholism described by Knight.[35] There was no correlation between the electroencephalogram and the presence or absence of psychogenic factors.

For 25 other patients the early influences were detrimental, but correlation with the development of personality was uncertain (case 2). The fathers of 12 patients showed rejection or hostility, and 10 of these were alcoholic and often cruel and abusive to their wives and children. Six fathers either died or deserted before the child reached 10 years of age. Three patients had hostile mothers and 5 lost their mothers before the age of 8 years. Five psychopaths were adopted from orphanages, between the ages of 1 and 4 years. In summation, only 15 of the 75 psychopaths had presumably normal backgrounds, and for 6 verified information about childhood was lacking. Factors such as these emphasize the inescapable importance of parent-child relationships in the development of the psychopathic personality syndrome.

SYNTHESIS

From the data presented it may be concluded that nearly all the psychopaths in this study were born with or acquired in infancy a defective cerebrum, a physicochemical abnormality (so far as can now be determined) which affects chiefly the frontal, or "silent," areas of the brain. Certainly there are quantitative and qualitative variations in this defect. In some extreme types of the psychopathic personality the result may be the organic hyperkinetic aggressiveness aptly described by Curran and Schilder[36] as the forerunner of the "hostile" psychopathic state. In other extreme types the cerebral organization may be so defective that minor emotional crises suffice to set off the dynamics of the psychopathic personality; here the psychogenic factors may not be apparent. However, not all persons who exhibit clinical evidence of damage to the brain have maladjusted personalities, and it is well known that 10 per cent of normal persons have abnormal brain waves. Similarly, many persons who encounter severe emotional trauma in childhood develop into presumably normal adults. In many psychopaths the cerebral dysfunction appears to increase the sensitivity to the emotional traumas of childhood. Unstable reactions to these traumas and inability to integrate new experiences into the growing personality further the development of psychopathic modes of behavior. This view is supported by the fact that every psychopath in this study showed either organic components or psychologically adverse factors. Depending, then, on the extent of cerebral dysfunction and on the adjustment level demanded of the personality, psychopathic behavior may first be manifested any time between infancy and early adulthood.

Since treatment of psychopaths along psychotherapeutic lines has not been fruitful, the knowledge that there exists an important organic component in their illness opens up new vistas for therapy. Experiments on the effects of various drugs and of electric shock are now in progress at the Medical Center.

---

35. Knight, R. P.: The Dynamics and Treatment of Chronic Alcohol Addiction, Bull. Menninger Clin. **1**:233-250, 1937.

36. Curran, F. J., and Schilder, P.: A Constructive Approach to the Problems of Childhood and Adolescence, J. Crim. Psychopath. **2**:125-152, 1940.

SUMMARY AND CONCLUSION

Seventy-five criminal psychopaths were studied both from an electroencephalographic and a clinical approach. The characteristics of the group were described. Electroencephalographic studies revealed 80 per cent abnormal or borderline abnormal tracings. Survey of the psychopath's developmental history showed that 80 per cent had psychologically unhealthy factors in childhood. From the available data the following conclusion seems warranted: Psychopathic personality is a mental illness resulting from inborn or early acquired cerebral dysfunction and disturbed parent-child relationships.

REPORT OF CASES

CASE 1.—*The excessively hostile psychopath.*

B. G., aged 18 years, is serving a minority sentence under the Federal Juvenile Delinquency Act for transportation of a stolen car across state lines. He was an unwanted and only child. His mother, aged 37, is a frivolous and narcissistic person who "thinks of nothing much except dressing up and running around." She gave her child little affection as an infant, and, in fact, left him mostly to the care of a meticulous and stern grandmother. His father, aged 42, makes a modest and adequate income as a mechanic. He has and had no interest in his child; he separated from his wife several times, drank to excess and often beat the patient with his fists for punishment. The patient's birth was considered normal. Early in infancy he manifested abnormal traits; he was overactive, nervous and demanding and displayed frequent temper tantrums. Later he became stubborn, quarrelsome, careless and bold. He frequently took money from his mother and grandmother, and later sundry things from school children; he became a facile liar, never admitted blame for his thefts and constructed fantastic tales to explain his ill gotten possessions. Progress at school was retarded; he had little ability to concentrate, played truant often and molested other children. Much of this school authorities blamed on parental neglect, for he usually was ill clothed and ill fed. He was placed in special vocational classes, without success, and achieved only a fifth grade standing at the age of 14 years. At that age he was first arrested for stealing articles from neighbors, among them a gun. During the next year he was arrested three more times for theft, twice for stealing guns. At the age of 16 he was sent to a state hospital for taking random shots at dogs, cats and cars. The diagnosis of psychopathic personality was then made, and he was discharged to the custody of two elderly, affectionate foster parents who had lost their own son. His behavior became more aggressive, and in the next six months he was arrested for theft of money, pistols and a car. He was given probation on each offense and was sent to an interested uncle in another city. There he fared no better. The uncle reported that he was unaffectionate, untrustworthy and irresponsible. Neighbors complained of missing articles, and he was suspected of molesting small girls. Within a month he fraudulently enlisted in the Army and four months later deserted. He was returned to the guardhouse but escaped by overpowering the guard. Then he hitch-hiked to his parents' home and received money, with which he was to travel to the nearest army post and turn himself over to the authorities. Instead, he went on a crime tour, which lasted two months. In that time he committed eleven armed robberies, thirteen automobile thefts, four burglaries and two attempts at burglary. Twice he shot at victims, seriously wounding 1 of them. He was captured only after being shot in the arm. He was sent to a parental home until trial, and there he beat a 58 year old guard to insensibility with a chair leg in an unsuccessful attempt to escape. He was then examined at a child guidance clinic. The psychiatrist found him mildly depressed over the prospect of imprisonment and noted hostility toward his father, an explosive temperament, vicious inclinations and poor tolerance to frustration. Neurologic examination revealed nothing abnormal, and his mental age on the Stanford-Binet test was 12 years and 10 months. The tentative diagnosis was psychopathic personality.

He was sent to a federal reformatory in February 1941, where he was placed in a special unit for psychopathic youths. Neurologic examination gave normal results, and he achieved a mental rating of 13 years on the Army alpha scale. There he acted like a child of 10, would not apply himself to tasks, disrupted the program, was impulsive and dangerous and had to be segregated. He was transferred to the Medical Center in July 1941. Examination showed him to be physically fit, though unattractive due to acne vulgaris and a heavy refractive error. Routine laboratory studies and neurologic examination revealed nothing abnormal. He seemed emotionally immature and at times childishly affable; he was without insight or

emotional attachments; he appeared intellectually dull and was unwilling to accept blame or to express remorse for his misdeeds. He admitted discord between his father and himself and said that if his father liked him "he sure doesn't show it."

At the Medical Center he exercises poor judgment and poor self control, demands attention for psychoneurotic or malingered complaints and often appears apathetic, indifferent, callous and hostile to persons in authority. Disciplinary violations continue, and once he viciously assaulted another inmate.

A special neurologic examination revealed the following abnormalities: slight flattening on the right side of the face, a stronger achilles reflex on the right side and a positive Hoffmann reflex on the left side. The electroencephalogram was of the "organic" type, with prominent 6 per second waves originating in the frontal region.

CASE 2.—*The excessively narcissistic psychopath.*

F. M., aged 25, is serving a sentence of three years for transportation of a stolen automobile across state lines (Dyer Act) and an additional two years for violation of parole on a prior sentence. His father died in World War I, when the patient was 1 year old. His mother, aged 40, is an inadequate person and had thrice been married. The first stepfather, a miner, provided fairly well for his family but "made life hard" for them; divorce occurred when the patient was 16 years of age. The patient bears the name of this stepfather and has 3 half-siblings, all of whom are well adjusted. His birth was considered normal. At an early age he was headstrong and wanted his own way. He became "absolutely a spoiled child," especially disobedient to his stepfather and hard to handle. He was overactive and stubborn, lied without compunction, was frequently a truant from school, took things that did not belong to him and fought with his siblings. In adolescence his family noted growing egotism, selfishness, carelessness and restlessness. He ran away from home several times and stayed away for weeks. Twice the juvenile court put him on probation for thefts, and at the age of 14 he was jailed three months for burglarizing box cars. The following year he was jailed six months for a similar offense. Attendance at school, which had been irregular, ceased at the age of 15, when he reached the ninth grade. He was "on the go," hitch-hiked around the country, did no work unless forced to and drank liquor excessively "for the kick." At the age of 17 he stole a car for a "joy ride" and was sent to a federal reformatory. There his maladjustment led to transfer to a penitentiary. On conditional release his behavior was no better. After less than a year of "freedom" he committed the current offense, another "joy ride."

He was sent to a federal penitentiary in July 1938. The psychiatrist made the diagnosis of unstable psychopathic personality. Soon his egotism became prominent and caused friction among his companions. On every work assignment he betrayed confidences and abused privileges. He usually had a superior attitude, tried to convince the authorities of his benign intentions and blamed others when he failed. He used institutional typewriters for illegal purposes, had government records photostated for his personal use and connived to get alcohol and drugs. For these offenses he lost all time for good behavior and was transferred to the Medical Center in January 1941.

On initial examination he was egotistic and evasive, claimed to have a poor memory or lied about his past record until he was confronted with the facts, when he blamed the officers, who "won't give him a break at all" and who were "always suspicious" of him. His thinking was superficial and his judgment defective. He had no close interpersonal attachments and was bitter about supposed maltreatment from his stepfather. He admitted having promiscuous sexual activities and was suspected of homosexual indulgences. His mental age was 18 years and 5 months on the Army alpha scale. Physical and neurologic examinations showed nothing abnormal except for a moderate tremor of the hands.

There has been no alteration of his fundamental characteristics. On every work assignment he soon adopts a superior attitude, causes friction with other inmates and spends his time in egocentric productions. For example, when he was typing literature on psychopathy he wrote a superficial "treatise" on its etiology. He makes numerous suggestions on how to better the institution, writes jingles, which he thinks should be published, and repeatedly submits to higher authorities sententious pleas for special consideration. He progressively has been demoted in work assignments, to which he reacts with projection thinking, sullenness and sometimes hypochondriasis.

A recheck of the neurologic examination revealed a positive resistance reflex on the left side and a fine tremor of the hand. The electroencephalogram was of borderline type, with general arrhythmia, asynchronism of the hemispheres and occasional 6 per second waves in the anterior region.

CASE 3.—*The excessively inadequate psychopath.*

A. F., aged 24, is serving a sentence of three years for transportation of a stolen automobile across state lines (Dyer Act). He was brought up in poverty-stricken circumstances. His

father, aged 50, was an unstable, improvident alcohol addict, who deserted his family when the patient was 3 years old. His mother, aged 46, was affectionate, did not remarry and struggled to raise her 3 children, of whom the patient is the youngest and the only delinquent. The patient's birth was considered normal; he had traumas to the head, with unconsciousness, at the ages of 1 and 19 years. As an infant he was nervous and hard to handle. As a child he was "spoiled" and quarreled and fought with other children. Early he started to lie, tell fantastic tales and steal from others. His mother punished him for these thefts, but he did not mind the strappings she gave him. His scholastic record was very poor; he advanced only to the second grade after seven years in school. He frequently was a truant, and several times he ran away from home. At the age of 10 he was brought before the juvenile court for petty thievery, and on the third arrest, at the age of 12, he was sent to a boarding home because he was thought too young to be sentenced to a reformatory. There he was maladjusted and ran away twice. At the age of 14 he stole a car and was committed to an industrial school for three years. After release he drank heavily, and in the next two years he was jailed four times for drunkenness and disorderly conduct. He changed jobs frequently, did little work and soon got into trouble by stealing another car. He spent two years in a state penitentiary, from which he attempted to escape. Then followed a year of unproductive "freedom," interrupted by a drunken brawl, in which he assulted another man. For this offense he was incarcerated two years in a state penitentary. On the day of his release he committed the current offense. His sister contended he "liked trouble" and that he had said he "would end in the electric chair."

At the federal penitentiary he impressed the psychiatrist as being an unstable, inadequate psychopath. He soon got into disciplinary difficulties for disobedience, loafing, leaving his detail without permission, carelessness about work, disorderly conduct, stealing food and having contraband in his possession. After eight months of maladjustment he was transferred to the Medical Center, in January 1941.

On initial examination he was affable and frank and freely admitted to his delinquencies. He expressed the belief that something was wrong with his mind, since he could not stop stealing and could not learn. He contended he had had heterosexual contacts since the age of 17, all of them with prostitutes. He never had any close interpersonal relationships but since childhood had been attached to his mother, and he said he would not consider marriage until after her death. On the Army alpha test his mental age was 15 years and 8 months. On the Wechsler-Bellevue test he achieved a performance rating of 15 years and 6 months, but verbal mental age was 9 years and 9 months. These uneven results suggested a cerebral pathologic disorder; special tests revealed severe reading and spelling disability and difficulty in hand dominance. Physical examination showed nothing abnormal except defective vision, and neurologic examination gave normal results.

His behavior continues to be unstable and inadequate. He cannot stand tedium and frequently requests changes in the ward or in his work. Often he makes a "sick call" for neurotic complaints. He is continually involved in petty violations of rules. His latest unprofitable escapade occurred one day before prospective conditional release; he stole a dozen watches from the storeroom and consequently lost all of his "good time." He could give no explanation for this and accepted punishment with equanimity.

Neurologic examination revealed only hyperactive tendon reflexes and a tendency to stutter. The electroencephalogram was normal (mixed type).

Dr. Howard L. Andrews, biophysicist at the United States Public Health Service Hospital, Lexington, Ky., gave help in the electroencephalographic technic and in the interpretation of the records.

Medical Center for Federal Prisoners.

INTEGRATED FACIAL PATTERNS ELICITED BY STIMULATION OF THE BRAIN STEM

EDWIN A. WEINSTEIN, M.D.
AND
MORRIS B. BENDER, M.D.
NEW YORK

Integration of the action of muscles innervated by the seventh cranial nerve has been of interest to neurophysiologists since the beginning of the nineteenth century. Sir Charles Bell [1] described "the marvelous combination of parts by which we have breathing, voice, speech, expression, smelling, coughing, sneezing and vomiting." Various levels and centers of the nervous system have been postulated for the selection of facial movements and their correlation with other somatic and autonomic components of purposeful acts. Sherrington [2] obtained contralateral contractions of facial muscles from cortical stimulation in the chimpanzee and the gorilla which were interpreted as entering into mimetic expression. From another portion of the cortical face area, contralateral movements of the lips suggesting feeding (chewing, deglutition) were elicited. Magoun, Ranson and Fisher [3] traced a pathway for rhythmic movements of the mouth and for tongue lapping from the cortex to the pons in the cat. Ectors, Brookens and Gerard [4] obtained facial and masticatory movements suggesting rage and fear from hypothalamic stimulation in cats. Kinnier Wilson,[5] on good clinicopathologic evidence, postulated a center in the brain stem at the line of the upper part of the pons linking the facial nucleus, the nucleus ambiguus and the respiratory centers. Sir Charles Bell expressed the belief that the peripheral nerve itself had separate fibers for expression, respiration and voluntary action.

In the present communication we are primarily concerned with the purposefully integrated facial movements obtained on electric stimulation of a discrete point in the brain stem. For this purpose the monkey is more suitable than the cat or the dog, not only in view of the development of its facial musculature but because of the closer functional and structural relation of its nervous system to that of man.

MATERIAL AND METHODS

Twenty-two monkeys (Macaca mulatta) were studied with the Horsley-Clarke stereotaxic apparatus according to the technic described by Ranson.[6] Stimulation of the diencephalon, midbrain, pons and medulla was carried out with a bipolar needle electrode in the vertical, diagonal

From the laboratories of the Mount Sinai Hospital.
This work has been aided by a grant from the Josiah Macy Jr. Foundation.

1. Bell, C.: The Nervous System of the Human Body, Washington, Duff Green, 1833.
2. Denny-Brown, D.: Selected Writings of Sir Charles Sherrington, London, Harper & Brothers, 1939, p. 412 and 414.
3. Magoun, H. W.; Ranson, S. W., and Fisher, C.: Corticofugal Pathways for Mastication, Lapping and Other Motor Functions in the Cat, Arch. Neurol. & Psychiat. **30**:292 (Aug.) 1933.
4. Ectors, L.; Brookens, N. L., and Gerard, R. W.: Autonomic and Motor Localization in the Hypothalamus, Arch. Neurol. & Psychiat. **39**:789 (April) 1938.
5. Wilson, S. A. K.: Modern Problems in Neurology, New York, William Wood & Company, 1929, chap. 12.
6. Ranson, S. W.: On the Use of the Horsley-Clarke Stereotaxic Instrument, Psychiat. en neurol. bl. **38**:534, 1934.

and horizontal planes of the stereotaxic instrument at 1 and 0.5 mm. intervals.[7] A pulsating, spike-shaped current was supplied through a Dumont variable frequency stimulator, the frequency being kept constant at 250 per second. The smallest electromotive force necessary to produce a minimal response was 12 volts, according to our instrument. This threshold was equivalent to 0.8 volt on the ordinary alternating, 60 cycle sinusoidal current. Every point explored was tested with varying strengths of current, but only the "primary" response, i. e., the response obtained with the minimal current, was considered as valid in the final analysis. The experiments were carried out with the animal under light anesthesia induced with soluble pentobarbital U. S. P. administered intravenously, the anesthesia being renewed during the procedure only when necessary, so that often we were able to stimulate the animal when it was practically awake. Observations were made on movements of the face, eyes, jaw, palate, epiglottis, tongue and limbs. Blood pressure readings were made on 4 animals. Gross changes in respiration were noted visually. The animals were killed with an overdose of soluble pentobarbital, and the brain stem was sectioned serially. The sections were stained by the Weil, hematoxylin and eosin and Nissl methods. In histologic determination of the exact points of stimulation, the vertical and horizontal needle tracks served as checks on each other. Allowance was made for changes due to shrinkage and retraction in pyroxylin embedding, these artefacts being particularly evident in the medulla.

RESULTS

Responses of facial muscles could be obtained by stimulating many areas of the midbrain, pons and medulla outside the nucleus of the facial nerve and its fibers. While many of these responses did not fit into any purposefully integrated function, certain coordinated patterns were recognized. These could be divided into three major types: (1) closure of the eyelids and straining; (2) sucking and swallowing, and (3) grimacing, suggestive of smiling or laughter.

Closure of the Eyelids.—This pattern consisted of contraction of the orbicularis oculi muscle on the same or on both sides. The contraction occurred more commonly bilaterally and when obtained at minimal voltage involved no other muscles innervated by the facial nerve. It was associated with bilateral upward rolling of the eyeballs (Bell's phenomenon) and on certain occasions by constriction of the pupils; this triad constitutes the lid closure reaction occurring normally in man and in other animals. A similar pattern of facial and ocular movements occurs in sleep when the lids close, the eyeballs roll up and slightly outward and the pupils constrict.

The response was obtained from the tegmentum of the pons 1.5 to 2.5 mm. from the midsagittal plane. In the vertical plane of the stereotaxic instrument, the pattern was elicited at anterior coordinates A + 2 to A + 5. This plane included the caudal part of the oculomotor nucleus, the medial longitudinal fasciculus, the decussation of the brachium conjunctivum, the medial lemniscus and the rostral border of the pyramids (fig. 1). The plane was rostral to the superior olive and the facial nucleus. Although they lay in the reticular substance, the reactive points appeared to be closely related to a lightly myelinated fiber bundle which has been identified by Papez as the reticulo-olivary tract, a component of the central tegmental fasciculus (*C. T. F.*). Stimulations made more ventrally and mesially resulted in bilateral action of the orbicularis oculi without ocular movement, while the more laterally situated reticular substance yielded pupillary dilatation. In still other adjacent areas unilateral closure of the lid and Bell's phenomenon were observed. Ingram, Ranson, Hannett, Zeiss and Terwilliger[8] observed contraction of the orbicularis oculi muscle on stimulating the central tegmental fasciculus in cats.

7. In expressing the coordinates of the stereotaxic instrument, we use the following symbols: A with a numeral indicates the number of millimeters rostrad (+) and caudad (—) to the zero frontal, or interaural, plane (A O), and H with a numeral, the number of millimeters dorsal (+) and ventral (—) to the zero horizontal plane (H O).

8. Ingram, W. R.; Ranson, S. W.; Hannett, F. I.; Zeiss, S. R., and Terwilliger, E. H.: Results of Stimulation of the Tegmentum with the Horsley-Clarke Stereotaxic Apparatus, Arch. Neurol. & Psychiat. **28**:513 (Sept.) 1932.

The phenomenon of lid closure occurring with slightly higher voltages was frequently accompanied by a peculiar pressing together of the lips, involving chiefly the quadratus labii muscles, and by closure of the jaws and cessation of respiration.

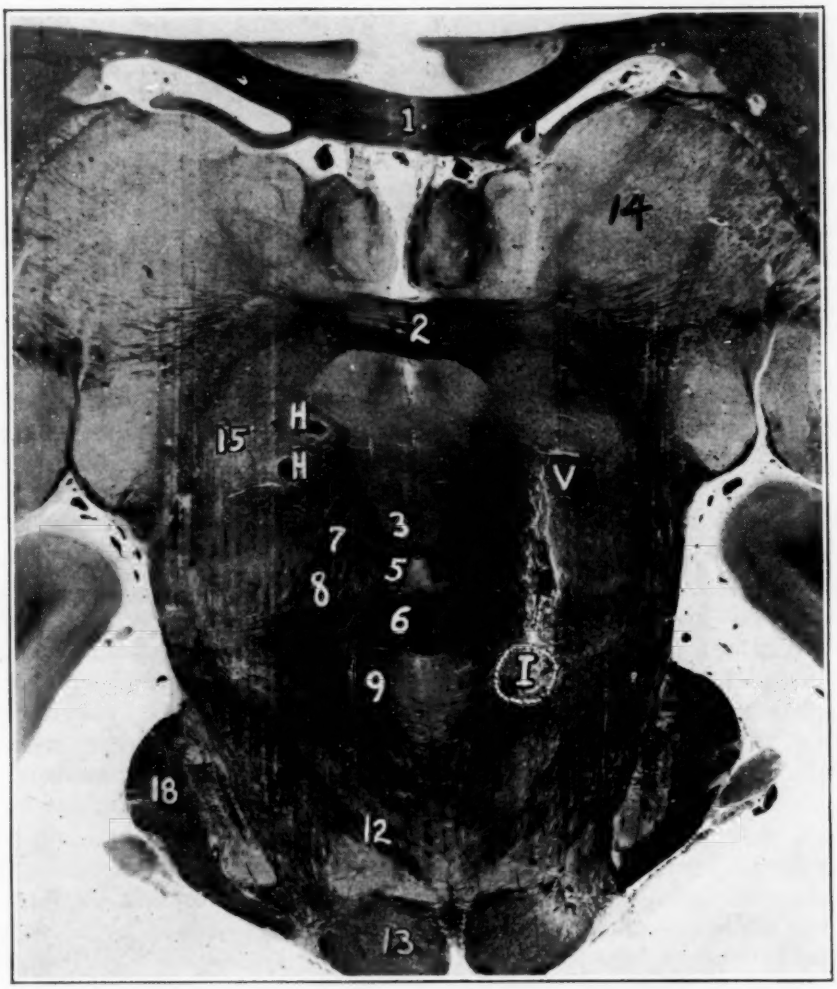


Fig. 1.—Frontal section of the brain stem of a monkey 3 to 5 mm. anterior to the zero frontal, or interaural, plane. The vertical needle track (*V*) is 2.8 mm. lateral to the midline and passes through the corpus callosum, the medial and dorsal thalamic nuclei, the posterior commissure, the pretectal area, the lateral border of the tecto-olivary and reticulo-olivary tracts, the brachium conjunctivum, the lateral portion of the reticulo-olivary tract, the reticular substance, the medial lemniscus and the pyramid. The best focus for the associated bilateral closure of the eyelids, upward rolling of the eyeballs (Bell's phenomenon) and pupillary constriction (eyelid closure reaction) is indicated by the white dash circle (*I*). This reactive area is located in the reticulo-olivary tract, which is part of the central tegmental fasciculus. Weil stain for myelin sheaths.

The structures in this section, and in the accompanying sections, of the brain stem may be identified by the following numbers and letters: *1*, corpus callosum; *2*, posterior commissure; *3*, oculomotor nucleus; *4*, trochlear nucleus; *5*, medial longitudinal fasciculus; *6*, brachium conjunctivum; *7*, thalamo-olivary (?) and tecto-olivary tracts; *8*, rubroreticulo-olivary tract (*7* and *8* making up the central tegmental fasciculus [*C. T. F.*]); *9*, reticulo-olivary tract; *10*, superior olive; *11*, inferior olive; *12*, medial lemniscus; *13*, pyramid; *14*, thalamus; *15*, pretectal area; *16*, superior colliculus; *17*, inferior colliculus; *18*, brachium pontis; *19*, nucleus of the abducens nerve; *20*, genu of the facial nerve; *21*, nucleus of the facial nerve; *22*, nucleus ambiguus; *23*, substantia nigra; *24*, red nucleus; *V*, vertical needle track, and *H*, horizontal needle track.

The compression of the lips, the tightly closed lids, the set jaws and the breath holding gave the monkey the appearance of partaking in some strenuous effort. This expression of straining was best obtained from the reticular substance of the pons at about the caudal borders of the responsive area for the lid closure reaction.

Sucking and Swallowing.—This pattern consisted of contraction of the orbicularis oris muscle, particularly of the upper lip, the response producing a pursing, sucking or nursing movement. This was constantly accompanied by (1) protrusion of the tip and elevation of the base of the tongue, with contraction of the geniohyoid and mylohyoid muscle; (2) elevation of the uvula and soft palate and (3) cessation of respiration in the inspiratory phase.

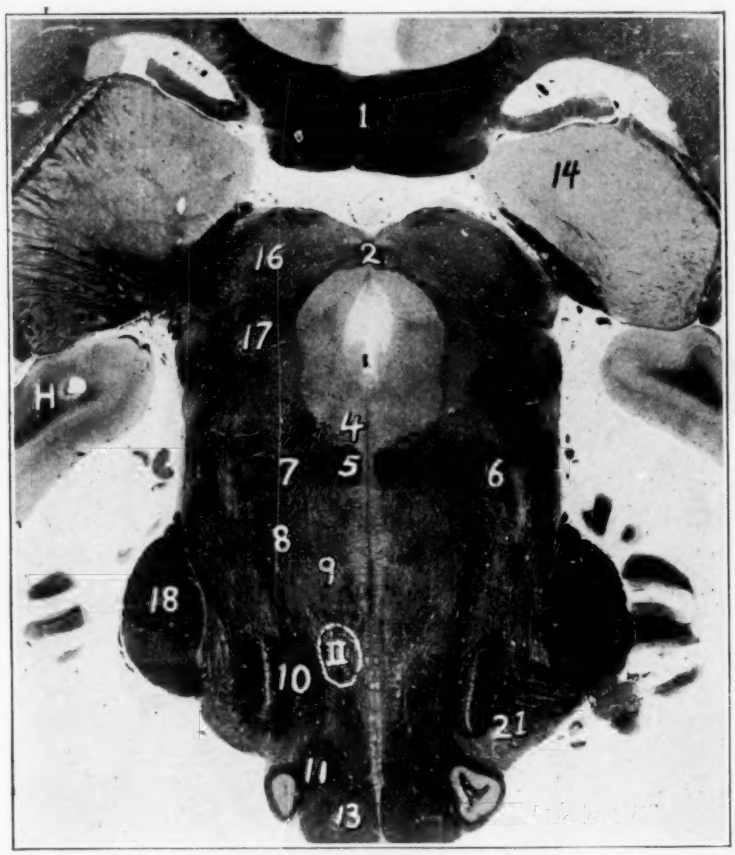


Fig. 2.—Frontal section of the brain stem of a monkey 1 mm. anterior to the zero frontal plane, illustrating the best reactive area for elicitation of sucking and swallowing movements (*II* within the white circle). Note the heavily myelinated rubrotecto-olivary tract and the lightly myelinated reticulo-olivary tract running toward the olive on the unnumbered side of the figure. Weil stain.

Frequently present were bilateral movements of the ears. On one occasion puffing out of the ipsilateral buccinator food pouch was observed. Contractions of the jaws were seen with stronger currents, but were not constant. A prompt, powerful bilateral closure of the jaws could be obtained from points in the midline of the tegmentum just ventral to the decussation of the brachium conjunctivum at planes H—7 and H—8. Salivation was occasionally seen. These complex coordinated movements suggested those involved in sucking or nursing and in the buccal

and pharyngeal phases of swallowing. In nursing, the nipple comes in contact chiefly with the orbicularis oris muscle of the upper lip. In deglutition, the mylohyoid muscles contract, so that the bolus is forced into the pharynx; the base of the tongue arches against the palate; the levator of the soft palate shuts off the posterior nares, and respiration is inhibited in the inspiratory phase that aspiration may be prevented. The movements of the ears are of interest; they are prominent as they

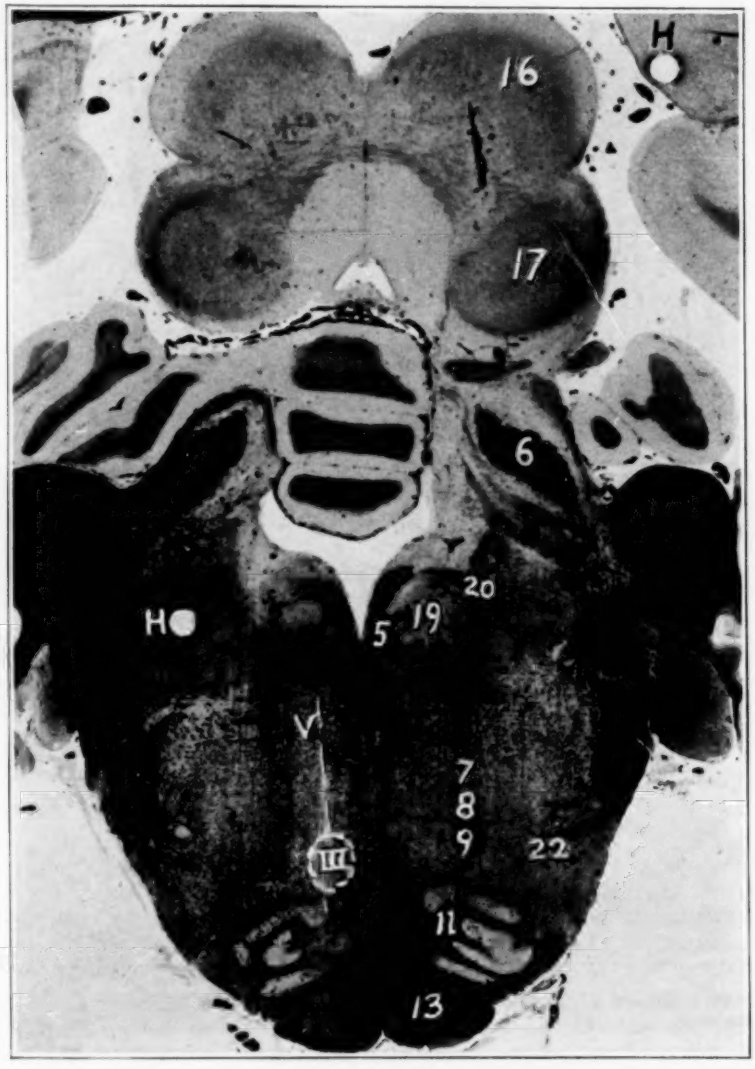


Fig. 3.—Frontal section of the brain stem of a monkey 2 mm. behind the zero frontal plane, illustrating the reactive area from which bilateral grimacing could be obtained (*III* within white circle). The vertical needle track (*V*) runs through the cerebellum, the genu of the facial nerve, the nucleus of the abducens nerve, the reticular substance, the mesial portion of the central tegmental fasciculus, the inferior olive and the pyramid. Weil stain for myelin sheaths.

are in the normal feeding macaque. Respiration was consistently inhibited at the end of inspiration no matter in what phase the stimulus was applied.

This pattern was obtained by stimulation 0.5 to 1.5 mm. from the midline in the tegmentum of the medulla dorsomesial to the rostral part of the inferior olive

(fig. 2). In the vertical plane of the stereotaxic instrument, the best results were elicited at A + 1 and A 0. The reactive area appeared to merge rostrally and laterally with the zones for straining and closure of the lids, as stimulation sometimes yielded fragments of each pattern (fig. 4). An entire complex, however, could be produced from a circumscribed field with the same minimal voltage, an indication that the various components of a pattern were not the result of spread

Fig. 4.—Sagittal section of the brain stem of a monkey 3 mm. lateral to the midplane. The areas which represent the best responses for bilateral and integrated facial movements are indicated by white dash circles. *I* is for closure of the eyelids and straining; *II*, for sucking and swallowing, and *III*, for grimacing. Weil stain for myelin sheaths.

from one reactive area to another. With greater voltage responses of the sixth nerve were obtained from the nearby intramedullary fibers of the nerve.

Grimacing.—In this movement the corners of the mouth were retracted and drawn up, the nostrils flared and the upper teeth were exposed to give the appearance of laughter or smiling. The response was part of a pattern which included

(1) depression of the lower jaw, with opening of the mouth; (2) retraction of the tip of the tongue, with lowering of the base; (3) depression and forward movement of the uvula, and (4) inhibition of respiration in the expiratory phase. Less consistent features were slight dilatation of the pupils, salivation and movements of the extremities. This pattern, like the sucking and swallowing complexes, was elicited by stimulations 0.5 to 2 mm. from the midsagittal plane, just dorsal to the olive and 2 to 3 mm. caudal to the reactive points for the swallowing complex at vertical coordinates A —3 to A —5 (fig. 3). Like the movements of the orbicularis oculi and the orbicularis oris muscles, the response was bilateral, static and smooth and persisted for the duration of the stimulation period. The respirations were consistently inhibited at the end of expiration no matter at which phase of respiration the current was applied.

This facial expression, simulating a hearty smile, has never been seen by us in the normal laboratory macaque, but has been observed in the chimpanzee in response to tickling. Darwin [9] cited several instances of a laughter grimace in tame monkeys. The expression differed strikingly from the snarling, baring of teeth and flattening back of the ears displayed by the enraged or frightened macaque. The tonic smiling or laughter pattern was also distinguished from the phasic, rhythmic grimacing, panting and vocalization and suggested a rage or pain reaction elicited experimentally by electrical stimulation from the region of the central gray matter, the inferior colliculus and the dorsolateral portion of the brain stem (Ferrier,[10] Thiele,[11] Magoun and Atlas, Ingersoll and Ranson [12]).

COMMENT

The patterns of facial movements just described which were observed on stimulation of circumscribed and focal areas in the brain stem with small currents are not simple reflex acts, but are associated, bilaterally integrated movements. Stimulation of the surrounding region or of any one of the cranial nerves or their nuclei fails to yield any of the complex reactions. Electrical exploration of the nucleus of the oculomotor, the trigeminal or the facial nerve does not produce the phenomenon of closure of the eyelid while neither the swallowing nor the "laughter" complex can be obtained from the nucleus ambiguus. The movements resulting from stimulation of the nucleus of the seventh nerve or its fibers are exclusively ipsilateral and tetanoid and involve only muscle groups innervated by the facial nerve, the action fitting into no purposeful scheme. The facial pattern movements elicited from the reactive areas, on the other hand, are bilateral, smooth and lifelike and are accompanied by actions of other cranial nerves and their autonomic components in a recognizably purposeful manner. Thus, in the sucking and swallowing complexes the action of the orbicularis oris muscle is associated with movements subserved by the fifth, tenth and twelfth pairs of cranial nerves, while in closure of the eyelids the contraction of the orbicularis oculi muscle is integrated with both the somatic and the autonomic components of each oculomotor nerve.

9. Darwin, C.: The Expression of Emotions in Man and Animals, New York, D. Appleton and Company, 1897, p. 132.

10. Ferrier, D.: The Functions of the Brain, London, Smith, Elder & Co., 1876.

11. Thiele, F. H.: On the Efferent Relationship of the Optic Thalamus and Deiters' Nucleus to the Spinal Cord, with Special Reference to the Cerebellar Influx Theory of Dr. Hughlings Jackson and the Genesis of the Decerebrate Rigidity of Ord and Sherrington, J. Physiol. **32:** 358, 1905.

12. Magoun, H. W.; Atlas, D.; Ingersoll, E. H., and Ranson, S. W.: Associated Facial, Vocal and Respiratory Components of Emotional Expression, J. Neurol. & Psychopath. **7:**241, 1936.

The facial movements are usually accompanied by alteration of respiration appropriate to the particular response. The nursing or swallowing pattern is associated with an arrest of respiration in the inspiratory phase, as in the normal act of preventing food from entering the trachea. The laughter-like grimace is the best example of the faciorespiratory synkinesia. The pulling up of the corners of the mouth, the flaring of the nostrils, the depression of the lower jaw, the retraction of the tongue and the lowering of the uvula are invariably associated with an arrest of respiration in the expiratory phase, such as occurs in laughter. On stimulating farther caudad in the medulla, close to the midsagittal plane, one may observe the facial and expiratory components of a coughing pattern. Another faciorespiratory correlation represented in the caudal portion of the medulla is a peculiar tensing grimace, accompanied by fixation of the thoracic and abdominal muscles and marked protrusion of the anus, as if the monkey were defecating. This confirms the division of the inspiratory-inhibitory and expiratory-inhibitory centers observed in the monkey by Beaton and Magoun.[13]

The facial patterns as they occur normally in the monkey and in man are both voluntary and involuntary. They resemble automatic, stereotyped movements, such as walking or swimming, which to a considerable degree are performed unconsciously. The question is where these facial movements are integrated in the nervous system. According to Ranson,[14] the subthalamus serves as a regulator and coordinator of such somatic functions. Direct stimulation of the subthalamus has indicated (Bard[15]; Hinsey[16]) that this structure is an integrating center for both the somatic and the visceral components of emotional expression, efferent paths making connections with the primary motor nuclei of the brain stem and the spinal cord. However, in our experiments stimulation of the monkey's hypothalamus failed to produce any coordinated facial expressions unless currents stronger than those previously described were used. Furthermore, there is no reason to believe that our responses from the brain stem were elicited from descending hypothalamic pathways. On the basis of our experiments, it seems that the brain stem is an important integrator of somatic functions of the cranial muscles.

The latter hypothesis is supported by the complicated and integrated bulbar acts that we[17] and others (Bazett[18]; Macht and Bard[19]) observed in decerebrate cats. With the transection made below the hypothalamus, facial expressions associated with swallowing, straining and anger in response to nocuous stimuli may be plainly visible. If the hypothalamus were preserved, it is probable that the facial expressions associated with emotional reactions would be more complex, and if still higher

13. Beaton, L. F., and Magoun, H. W.: Localization of the Medullary Respiratory Centers in the Cat, Am. J. Physiol. **126**:673, 1939.

14. Ranson, S. W.: The Anatomy of the Nervous System, Philadelphia, W. B. Saunders Company, 1943, p. 216.

15. Bard, P.: The Neuro-Humoral Basis of Emotional Reactions, in Murchison, C. A.: A Hand Book for General Experimental Psychology, Worcester, Mass., Clark University Press, 1934, chap. 6, p. 264.

16. Hinsey, J. C.: The Hypothalamus and Somatic Response, A. Research Nerv. & Ment. Dis., Proc. (1939) **20**:657, 1940.

17. Six decerebrate cats were studied in the laboratories of experimental neurology at New York University College of Medicine. In 1 of these animals, which lived one hundred and forty-six days, the brain stem was transected below the level of the oculomotor nucleus by Drs. L. Goodman and B. S. Brody, of Yale University.

18. Bazett, H. C.: A Study of the Sherrington Decerebrate Animal in the Chronic as Well as the Acute Condition, Brain **45**:185, 1922.

19. Macht, M. B., and Bard, P.: Studies on Decerebrate Cats in the Chronic State, Proc. Federation Am. Soc. Exper. Biol. **1**:55, 1942.

centers were intact the facial movements would be still more elaborate and varied. We believe that the brain stem is the ultimate integrating center for facial expression.

In considering the exact anatomic localization of the facial pattern, it is difficult to determine whether the nuclei in the reticular formation or the tracts traversing its substance are responsible for the effect of stimulation. The reactive areas appear to be in the region of the rubroreticulo-olivary system described by Papez.[20] This tract arises from the nucleus of Forel's field H and the red nucleus, descends in the reticular formation and makes connections with the various reticular nuclei and the ipsilateral inferior olive. Ogawa [21] has traced a similar path in the cat—a medial tegmental tract originating in the nuclei of Darkshevich, Cajal and Forel's field H and descending close to the midline of the tegmentum to enter the inferior olive. Papez [20] and Krieg [22] pointed out that the reticulo-olivary system is not continuous, but makes frequent synapses with the reticular nuclei of the pons and medulla. This may account for the changing patterns evolved at different levels as facial components are successively correlated in the brain stem with various types of somatic and respiratory actions.

SUMMARY

Stimulation of designated areas in the tegmentum of the brain stem of the macaque monkey with the Horsley-Clarke stereotaxic technic produces facial patterns integrated with other somatic and autonomic components into purposeful acts.

The facio-ocular synkinesis of contraction of the orbicularis oculi muscles, upward rolling of the eyeballs and constriction of the pupils can be elicited from the reticular substance of the pons 1.5 to 2.5 mm. lateral to the midsagittal plane.

Contraction of the orbicularis oris muscle in a sucking, swallowing movement is associated with elevation of the base of the tongue, raising of the uvula and inhibition of respiration in the inspiratory phase. This pattern is elicited from the reticular formation of the medulla 0.5 to 1.5 mm. from the midsagittal plane dorsomedial to the rostral part of the inferior olive.

A faciorespiratory complex simulating laughter and consisting of retraction and elevation of the corners of the mouth, depression of the lower jaw, lowering of the base of the tongue and uvula and cessation of respiration in the expiratory phase can be elicited from an area 0.5 to 2 mm. from the midsagittal plane dorsomesial to the inferior olive.

It is suggested that the facio-ocular and faciorespiratory synkinesias are integrated in the reticular formation of the brain stem.

Dr. J. W. Papez, of Cornell University, Ithaca, N. Y., aided in the identification of some of the anatomic structures in the brain stem of the monkey.

Mount Sinai Hospital.

20. Papez, J. W.: Comparative Neurology, New York, Thomas Y. Crowell Company, 1929. Papez, J. W., and Stotler, W. A.: Connections of the Red Nucleus, Arch. Neurol. & Psychiat. **44**:776 (Oct.) 1940.

21. Ogawa, T.: The Tractus Tegmenti Medialis and Its Connection with the Inferior Olive in the Cat, J. Comp. Neurol. **70**:181, 1939.

22. Krieg, W. J. S.: Functional Neuroanatomy, Philadelphia, The Blakiston Company, 1942.

PRIMARY SARCOMAS OF THE BRAIN

REVIEW OF THE LITERATURE AND REPORT OF TWELVE CASES

KENNETH H. ABBOTT, M.D.
Fellow in Neurosurgery, the Mayo Foundation
AND
JAMES W. KERNOHAN, M.D.
ROCHESTER, MINN.

Virchow [1] established the identity of the intracranial tumors arising from the supporting glial elements and named them "gliomas." Prior to his work their identity had been obscure and controversial, but, as a matter of fact, the differential classification was not successfully achieved until more than sixty years after Virchow's investigation, when Bailey [2] and Bailey and Cushing [3] correlated the histologic and the clinical aspects of the tumors. During these six decades glial and mesodermal tumors continued to be confused, as is evidenced by such misleading terms as "gliosarcoma." The existence of this state of confusion is not surprising when it is considered that up to the time of Bailey and Cushing [3] the number of brain tumors which observers had at their disposal was not sufficient to enable them to study these neoplasms properly. After Bailey's [4] excellent definition, in 1929, of primary sarcomas of the brain, reports of such tumors, with few exceptions, disappeared from the literature. The exceptions are notably such papers as those of Környey,[5] Mage and Scherer [6] and Hsü,[7] among others.[8] In view of the paucity of such studies, there appears to be room in the literature for another review of primary sarcomas of the brain, for it is only by the accumulation of a sufficiently large number of these rare neoplasms that a more complete understanding of them can be achieved.

From the Section on Pathologic Anatomy (Dr. Kernohan) and the Section on Neurosurgery (Dr. Abbott), the Mayo Clinic.

Abridgment of a thesis submitted to the faculty of the Graduate School of the University of Minnesota in partial fulfilment of the requirements for the degree of Master of Science in Neurosurgery.

1. Virchow, R.: Die krankhaften Geschwülste, Berlin, A. Hirschwald, 1864-1865, vol. 2, pp. 129-151.

2. Bailey, P.: Histologic Atlas of Gliomas, Arch. Path. **4**:871-921 (Dec.) 1927.

3. Bailey, P., and Cushing, H.: A Classification of the Tumors of the Glioma Group on a Histogenetic Basis with a Correlated Study of Prognosis, Philadelphia, J. B. Lippincott Company, 1926.

4. Bailey, P.: Intracranial Sarcomatous Tumors of Leptomeningeal Origin, Arch. Surg. **18**:1359-1402 (April) 1929.

5. Környey, S.: Eine sich entlang den Gefässwandungen ausbreitende Hirngeschwulst (adventitielles Sarkom); zugleich Bemerkungen zur Frage des Perithelioms des Gehirns, Ztschr. f. d. ges. Neurol. u. Psychiat. **149**:50-67, 1933.

6. Mage, J., and Scherer, H. J.: Tumeur cérébrale parvicellulaire se propageant dans l'espace de Virchow-Robin (la question des sarcomes adventitiels périvasculaires ou périthéliaux), J. belge de neurol. et de psychiat. **37**:731-746 (Dec.) 1937.

7. Hsü, Y. K.: Primary Intracranial Sarcomas, Arch. Neurol. & Psychiat. **43**:901-924 (May) 1940.

8. Bailey, P., and Bucy, P. C.: The Origin and Nature of Meningeal Tumors, Am. J. Cancer **15**:15-54 (Jan.) 1931.

AIM AND SCOPE

This study is therefore concerned with those primary sarcomas [9] which have arisen within the substance of the brain and have therein formed a tumor mass or have spread diffusely through the tissue of the central nervous system. The study is not concerned with hemangioblastoma or with sarcoma of the meninges.

For the most part, reference to the literature prior to the publication of Bailey and Cushing's [3] classification of the gliomas, in 1926, has been purposely omitted, except in a few instances, since it has been shown by one of us (Abbott [10]) that it is impossible to determine the nature of the tumors so frequently and erroneously described as "sarcoma" and masked under various modifying terms, such as "angiosarcoma," "gliosarcoma," "small round cell sarcoma," "meningiomatosis," "endotheliomatosis," "perithelioma" and "periendothelioma." The literature published since 1926 contains only an occasional reference to primary sarcoma of the brain; in fact, so few are such references that one is led to believe either that the tumor actually is rare or that pathologists are reluctant to admit such a diagnosis. It is probable that both these factors operate to explain the situation. Hence, a review of the problem seemed justified.

Definite criteria for the recognition of sarcomas throughout the body seem to be well established and understood; therefore, we have undertaken to review these criteria as they apply to primary mesodermal tumors of the brain and to classify and report such tumors recorded in the files of the Mayo Clinic.

MATERIAL

Search of the records of the Mayo Clinic which concern tumors of the brain yielded 12 cases of neoplasms which could be considered to come within the confines of this survey. Another neoplasm, a surgical specimen removed for biopsy, probably belonged to this group, but consisted of insufficient tissue for us to be able to prove its identity beyond a doubt. It is with these 12 tumors that we are concerned herein.

HISTOLOGIC, EMBRYOLOGIC AND CLINICAL CONSIDERATIONS

Embryologic Aspects.—In consideration of the tissue or tissues from which primary sarcomas of the brain arise, the problem of the cerebral vessels and the pia mater is of immediate importance. However, it is not within the province of this paper to survey in detail the anatomic structure or the embryonic origin of the tissues forming the boundaries of the Virchow-Robin space [11]; on the contrary, it seems enough for the purposes of this study to recognize that connective tissue exists in the adventitia of the intracranial blood vessels and in other parts of their

9. Sarcoma is here considered as a tumor composed of cells of the connective tissue type; the older definitions of its origin from the mesoderm cannot be accepted in the light of present day embryologic considerations. It is probably safe to say that no embryologist any longer clings to the outmoded theory of specificity of the germ layers. Therefore, in defining sarcoma it seems correct to characterize it as derived from, or as morphologically reproducing, connective tissue.

10. Abbott, K. H.: Primary Sarcomas of the Brain and a Report of Two Cases of Primary Lymphosarcoma of the Intracranial Dura Mater, Thesis, University of Minnesota Graduate School, 1942.

11. (*a*) Eberth, C. J.: Ueber die Blut und Lymphgefässe des Gehirns und Rückenmarks, Virchows Arch. f. path. Anat. **49**:48-50 (Dec. 11) 1870. (*b*) Schaltenbrand, G., and Bailey, P.: Die perivaskuläre Piagliamembran des Gehirns, J. f. Psychol. u. Neurol. **35**:199-278 (March) 1928.

walls. It is recognized, likewise, that although Harvey and Burr,[12] Harvey, Burr and Van Campenhout [13] and, more recently, Raven [14] have presented evidence which is sufficient to cast doubt on the mesodermal theory of origin of the leptomeninges and to support the theory of their ectodermal derivation, these envelopes, so to speak, of the nervous system apparently do reproduce tumors of the nature of connective tissue. This is well borne out by the fact that the meningiomatous group of tumors is now considered of leptomeningeal origin.[15]

Although space does not permit here a detailed discussion of the possibility that certain tumors arise from the microglia, it would seem, on the basis of the work of del Río-Hortega and Jimenez de Asúa [16] on the silver impregnation of both microglia and macroglia and of Penfield's [17] studies, that all the tumors under discussion are either sarcomas (Yuile [18]) or gliomas containing many microglia cells (Benedek and Juba,[19] Awzen [20]).

Histologic Characteristics.—The group of sarcomas considered herein were characterized by the formation of tumor masses and by local and perivascular extension of the neoplasm. No part of the brain could be considered as a site of predilection. None of the tumors studied metastasized to regions outside the central nervous system. Some of the tumors within the substance of the brain were enveloped by a pseudocapsule of degenerative nerve tissue, but in no instance was a true capsule present around the tumor mass. Until such a tumor situated in the cerebrum was studied recently, we had seen this type only in the cerebellum. On cut section tumors of this type had a more or less soft, "fleshy" appearance, as the term "sarcoma" ("resembling flesh") was originally interpreted to indicate. Although perivascular spreading was seen histologically, it was not so prominent as in the tumors characterized by generalized, diffuse perivascular sarcomatosis of the brain, such as has been reported by other authors.[21] In tumors of the latter type, in which grossly diffuse characteristics were presented, local tumefaction could be seen microscopically to be the coalescence of a tumor which probably had begun as a perivascular neoplasm, with "breaking through" of the cells from

12. Harvey, S. C., and Burr, H. S.: The Development of the Meninges, Arch. Neurol. & Psychiat. **15**:545-565 (May) 1926.

13. Harvey, S. C.; Burr, H. S., and Van Campenhout, E.: Development of the Meninges: Further Experiments, Arch. Neurol. & Psychiat. **29**:683-690 (April) 1933.

14. Raven, C. P.: Zur Entwicklung der Ganglienleiste: V. Ueber die Differenzierung des Rumpfganglienleistenmaterials, Arch. f. Entwcklngsmechn. d. Organ. **134**:122-146 (March 9) 1936.

15. This is particularly true of the intraventricular meningiomas, which Abbott and Courville [24] have recently shown to be composed entirely of fibrous connective tissue.

16. del Río-Hortega, P., and Jimenez de Asúa, F.: Sobre la fagocitosis en los tumores y en otros procesos patológicos, Arch. cardiol. y hemat. **2**:161-220, 1921.

17. Penfield, W.: The Encapsulated Tumors of the Nervous System: Meningeal Fibroblastomata, Perineurial Fibroblastomata and Neurofibromata of von Recklinghausen, Surg., Gynec. & Obst. **45**:178-188 (Aug.) 1927.

18. Yuile, C. L.: Case of Primary Reticulum Cell Sarcoma of the Brain: Relationship of Microglia Cells to Histiocytes, Arch. Path. **26**:1036-1044 (Nov.) 1938.

19. Benedek, L., and Juba, A.: Microglioma, Gyógyászat **81**:223-226 (April 19) 1941.

20. Awzen, A. P.: Du type spécial des tumeurs mésenchymes non mûries du système nerveux central: (un cas de mésoglioblastoma), Acta med. Scandinav. **87**:470-486, 1936.

21. (*a*) Fried, B. M.: Sarcomatosis of the Brain, Arch. Neurol. & Psychiat. **15**:205-217 (Feb.) 1926. (*b*) Hassin, G. B.: Histopathology of the Peripheral and Central Nervous Systems, ed. 2, New York, Paul B. Hoeber, Inc., 1940, p. 467. (*c*) Környey.[5] (*d*) Mage and Scherer.[6]

the Virchow-Robin space, so that the nerve tissue situated between the vessels had been invaded and an ill defined tumor mass had been formed. Two tumors of our series somewhat simulated tumors of this type: In 1 of them the spreading was extensive, having involved even the spinal cord; the other tumor apparently had originated in the ventricles and spread diffusely into the proximal neural tissues. Other tumors in this series were seen to replace large portions of nerve tissue by invasion and subsequent replacement of necrotic nerve tissue by their tumor structure; this was particularly characteristic of the fibrosarcoma.

Clinical Aspects.—Clinically, these primary sarcomas of the brain occurred among patients ranging in age from 4 to 57 years. The patients were distributed according to decades of life, as follows: One was in the first decade; 2 were in the second; 1 was in the third; 3 were in the fourth; 4 were in the fifth, and 1 was in the sixth. The average age was 31.6 years. Thus, the tumors tended to occur among fairly young adults, about half (5) of the patients being between the ages of 20 and 40 years and all except 1 patient being less than 50 years of age.

The tumors considered herein varied clinically from slowly to rapidly growing malignant neoplasms, occurring in all parts of the brain; their symptomatic aspects thus varied according to the factors of both rate of growth and situation.

MAIN TYPES OF SARCOMA OF THE BRAIN

Microscopically, primary sarcomas of the brain can be divided into two, or possibly three, histologic types, namely: (1) fibrosarcoma; (2) perivascular sarcoma ("perithelial" or "adventitial" sarcoma), and (3) sarcoma of unknown type. Sarcoma of the brain as encountered in this study will be considered under these three headings.

FIBROSARCOMA

In this series of 12 tumors there were 3 primary intracerebral fibrosarcomas. Another tumor classified as sarcoma of unknown type probably belongs in this group, since we have tentatively classified it as a "malignant giant cell fibrosarcoma." Since the degree of malignancy of the tumors in this group varied so greatly, a description of 1 fibrosarcoma of a low degree and 1 of a high degree of malignancy will be presented.

CASE 1.—*Typical fibrosarcoma of the left lateral ventricle and cerebral hemisphere.*

The patient, a girl 4 years old (case 1, table 1), was the youngest in the entire series. Intermittent attacks of vomiting had occurred for one year. Two and a half months before her entry into the clinic she had suffered from mild otitis media on the right side. For four weeks she had had bouts of sudden occipital pain followed by vomiting. Recently, progressive hemiparesis had developed on the right side. Examination, on March 12, 1937, revealed evidence of a left frontotemporal lesion. Subtemporal exploration and decompression were performed, but a tumor was not encountered. The patient grew progressively worse and died at home on May 12, 1937.

Necropsy, performed at the patient's home, revealed a soft, well circumscribed tumor, measuring 5 by 5 by 6 cm. The neoplasm apparently arose from the medial and posterior walls of the left lateral ventricle. It had expanded laterally, practically filling the ventricle, which had undergone considerable pressure atrophy. Blocks of tissue, forwarded for study, revealed a rather typical, slow-growing fibrosarcoma (fig. 1), containing interlacing bundles of slender and fat spindle cells, collagen and reticulin. Occasional mitoses were present. The meninges in the left frontotemporal region were involved, as shown by heavy subarachnoid invasion by the tumor cells, which in places had totally obliterated the leptomeningeal structures. In several adjacent portions the leptomeninges were infiltrated with

tumor cells, which extended for a short distance into the underlying cortex. Because we did not have blocks of tissue from other regions of the brain, it is not possible to state the extent of the leptomeningeal spread of the tumor.

The diagnosis was fibrosarcoma, of well differentiated type.

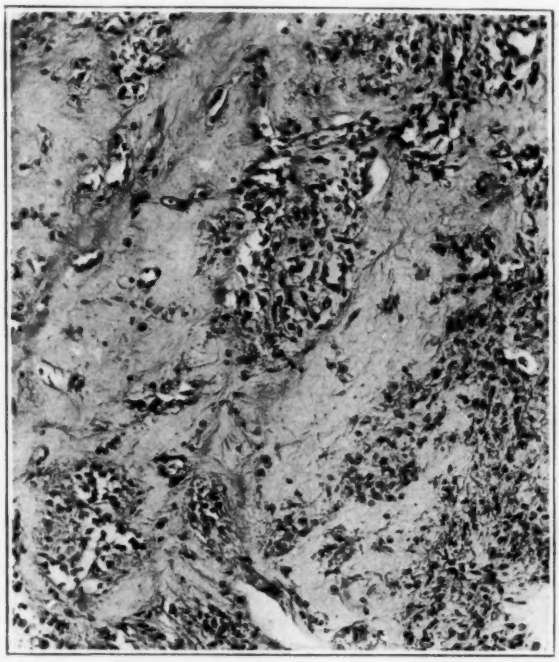


Fig. 1 (case 1).—A slowly growing fibrosarcoma, showing an unusual amount of collagenous stroma, scattered about in which are active sarcoma cells. Hematoxylin and eosin; × 110.

TABLE 1.—*Clinicopathologic Data on Twelve Cases of Primary Sarcoma of the Brain*

Patient			Operation	Duration of Symptoms *	Post-operative Survival	Situation of Tumor	Involvement of	
No.	Age, Yr.	Sex					Meninges	Ventricles
				Fibrosarcoma				
1	4	F	Yes	1 yr.	2 mo.	Right cerebral hemisphere	Yes	Yes (origin in ventricle [?])
2	21	M	No	8 wk.	†	Left occipital lobe	Yes	No
3	14	F	Yes	6 yr.	7 days	Right temporal lobe	Yes	No
				Perivascular Sarcoma				
4	17	M	Yes	6 wk.	13 hr.	Right cerebellar hemisphere	No	No
5	57	M	No	6 wk.	†	Right lateral and fourth ventricles	Yes	Yes
6	41	M	No	11 wk.	†	Right cerebral hemisphere	Yes	No
7	34	M	Yes	3 mo.	2 days	Left cerebellar hemisphere	Yes	No
8	49	M	Yes	5 mo.	3 days	Left frontoparietal lobe	No	No
9	38	M	Yes	2 mo.	5+ mo.	Left cerebellar hemisphere	?	?
10	48	M	Yes	5 wk.	4½ mo.	Right temporal lobe	?	?
				Sarcoma of Unknown Type				
11	34	F	No	9 mo.	†	Left frontotemporal region	Yes	Yes
12	40	M	Yes	3 mo.	3+ yr.	Left temporoparietal region	Yes	?

* Until the time of operation or of admission to the clinic.
† These patients did not undergo operation. Survival rates for such patients may be found in the text.

CASE 2.—*Subcortical fibrosarcoma of the left occipital lobe.*

A 21 year old man (case 2, table 1) was first examined on Nov. 4, 1933. The onset of his condition, eight weeks previously, had been characterized by ataxia, vomiting of projectile type and frontal headache. For six weeks he had experienced intermittent paresthesia and paresis of his right hand and fingers. He had had vertigo and diminution of vision for one month, followed by episodes of fainting and generalized convulsions and tinnitus, with pain in both ears. Results of the general physical examination were not abnormal, but neurologic inspection revealed bilaterally choked optic disks, with retinal hemorrhages and exudates and homonymous hemianopia in the right eye. The patient died rather suddenly, during a sudden paroxysm of vascular hypertension, one day after his admission.

Necropsy, carried out soon after his death, revealed chronic tuberculosis of the spleen and the hilar nodes of the lungs, mild aortic sclerosis and a tumor of the left occipital lobe. After fixation of the brain, a whitish tumor, firm in consistency and measuring 3 by 4 cm., was observed in the left occipital lobe, surrounded by a small zone of softened brain tissue.

Microscopic study of the sections (fig. 2 *a*, *b* and *c*) disclosed a fairly cellular tumor, with wide variations in differentiation. The appearance was that of an ordinary fibrosarcoma,

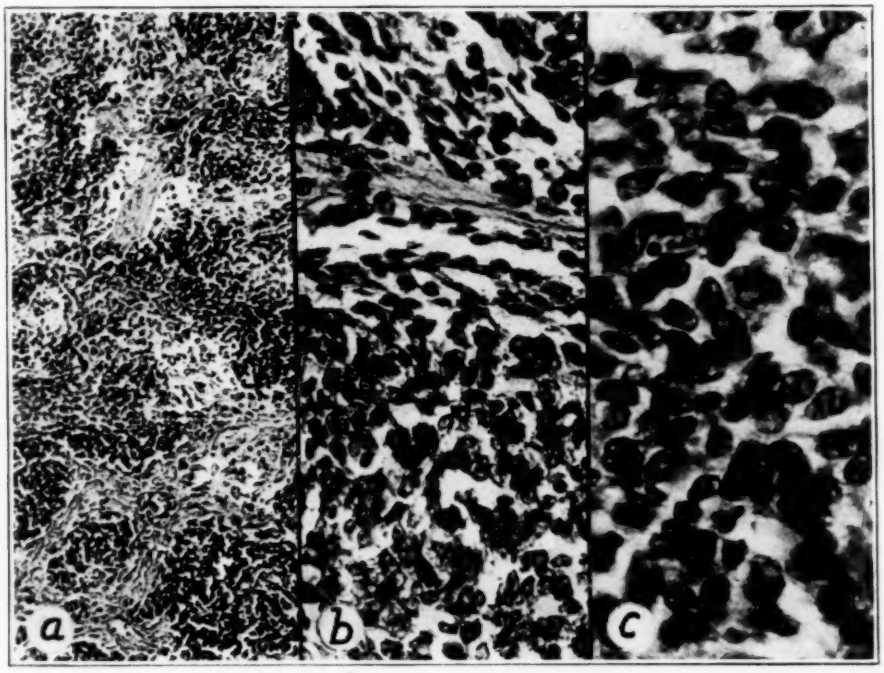


Fig. 2 (case 2).—Fibrosarcoma: (*a*) Irregular nests of cells ("pseudoalveolar" nests) in a fibrous stroma (× 100); (*b*) adjacent nests of cells, separated by a thin band of fibrous and reticulin stroma (× 325); (*c*) high power view of the malignant sarcoma cells seen in the nests (× 650). Compare the cells shown in *c* with those of the perivascular sarcoma seen in figure 4 *a* and *b*. Hematoxylin and eosin stain.

with irregular islands, or "alveoli," filled with closely packed, less differentiated spindle and oval nuclei surrounded by a small amount of cytoplasm (fig. 2 *b*). Many mitotic figures were present. These areas were surrounded by spindle cells, collagen and reticulin, in the center of which small blood vessels were frequently, but not always, encountered. Thus, as is shown in figure 2 *a*, a "pseudoalveolar" arrangement was present. The malignant sarcoma cells contained in the nests are seen under high magnification in figure 2 *c*.

Blood vessels of various sizes were numerous throughout the sections of the neoplasm. The larger vessels frequently were surrounded by layers of tumor cells and collagen, so that they appeared to have very thick walls.

The nerve tissue exhibited various degrees of necrosis and acute and subacute degeneration. Not infrequently the vessels in the degenerated tissue, as well as in the fairly normal tissue, were surrounded by cuffs of tumor cells. Sections of the meninges did not contain any evidence of meningeal extension of the tumor.

The diagnosis was fibrosarcoma, of highly malignant type.

CASE 3.—*Fibrosarcoma of the right temporal lobe.*

A girl aged 14 years (case 3, table 1) had experienced epileptic seizures for six years. During the three years previous to her admission there had been progressive enlargement of the head. Aside from evidence of prolonged increase in the intracranial pressure, there were no localizing signs. Suboccipital exploration did not reveal the causative lesion, and the patient died one week after operation.

Necropsy disclosed a large fibrosarcoma of the right temporal lobe, with small implants of the tumor in the leptomeninges over the cerebellum and the upper cervical portion of the spinal cord. In some areas there was a striking tendency toward perivascular growth. The appearance of such an arrangement was accentuated by the presence of intervascular necrosis (fig. 3*a*; this section should be compared with the section from the perivascular sarcoma in case 9, fig. 3*b*).

Of the 12 primary sarcomas of the brain constituting the basis of this report, 3, or one fourth, were intracerebral fibrosarcomas; but the series is probably

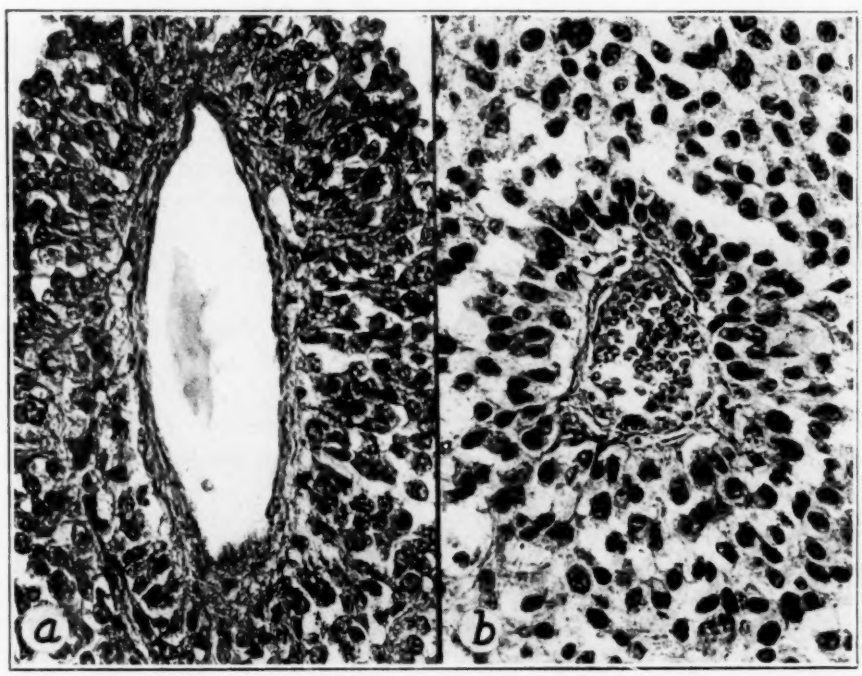


Fig. 3.—(*a*) Fibrosarcoma in case 3 (× 250): The appearance of perivascular growth in this area was aided by the presence of intervascular necrosis. (*b*) Perivascular sarcoma in case 9 (× 295): The radial perivascular growth of this tumor, with its cells of more delicate internal architecture, is to be compared with that in the fibrosarcoma shown in *a*. Hematoxylin and eosin stain.

too small for this fact to be of significance. However, among the cases of primary sarcoma of the brain reported in the literature, again in about one fourth the tumors were intracerebral fibrosarcomas. The incidence according to age in the 3 cases just reported would lead to the belief that fibrosarcomas occur almost exclusively among children and young adults, whereas in a review of the literature we found that the ages of the patients ranged from 17 months to 63 years. When we arranged these cases according to decades of life, a fairly uniform spread occurred up to the fifth decade, in which the largest number of cases was found.

Because of insufficient data, the survival periods of the patients in this group of our series cannot be accurately stated, but they appear to have ranged from two months to six years, as determined by the date of onset of symptoms to the date of the patient's death. The period of survival obviously depends on several

factors, among which are the degree of biologic activity (malignancy) of the neoplasm, the situation of the tumor, the ability of the cranial sutures to separate, and thus to allow a degree of decompression, and, probably least important of all, the operative intervention. On the whole, patients in this group (with fibrosarcoma) lived considerably longer than those in the next group, a fact which suggests that fibrosarcoma has a lower degree of malignancy than other tumors of the brain. An exception is seen in the second case, in which the tumor appeared

TABLE 2.—*Reported Cases of Fibrosarcoma and Fibroblastoma of the Brain*

Author	Year	Location of Tumor	Age of Patient, Yr.	Nomenclature
Houlton [23b]	1926	Two tumors: genu of left internal capsule and subcortex of left fontal lobe	47	Multiple sarcoma
Zagni (Néoplasmes **5**: 159-173, 1926)..	1926	Centrum of right frontoparietal area	40	Sarcome primitif
Zagni (Néoplasmes **5**: 159-173, 1926)..	1926	Centrum of left parieto-occipital region	44	Sarcome primitif
Bailey [4]	1929	Right temporal region	42	Fibrosarcoma
Bailey [4] (Mallory's case)	1929	Right frontal region	33	Fibrosarcoma
Bailey [4]	1929	Right lateral ventricle	19	Fibrosarcoma
Armenise (Cervello **11**: 25-39 [Jan. 15] 1932)	1932	Right temporosphenoid lobe	63	Fibrosarcoma
Alpers and others (Arch. Neurol. & Psychiat. **27**: 270-281 [Feb.] 1932)	1932	Right frontotemporal lobe	52	Fibroblastoma
Foot and Cohen [44]	1933	Left temporoparietal subcortex	9	Retotheliosarcoma
Meyer and Scheller (Virchows Arch. f. path. Anat. **300**: 473-486 [Aug. 6] 1937)	1937	Left temporal region	25	Fibromyxoma
Baker and Adams (Am. J. Path. **13**: 129-138 [Jan.] 1937)	1937	Right frontal region	10	Fibroblastoma
Ferens (Neurol. polska **21**: 387-402, 1938)	1938	Cerebral centrum	36	Primary reticuloendothelioma
Balduzzi [45]	1938	Cerebral centrum	41	Sarcoma magnocellulare
Cottrell [23a]	1939	Two tumors: left frontal and left temporal regions	52	Fibrosarcoma
Bailey, Buchanan and Bucy [25] *	1939 Case 22	Vermis cerebelli	8	Alveolar sarcoma
Bailey, Buchanan and Bucy [25]	Case 23	Vermis cerebelli, with widespread cerebrospinal implants	11	Alveolar sarcoma
Bailey, Buchanan and Bucy [25]	Case 26	Vermis cerebelli	17 mo.	Alveolar sarcoma
Hsü [7]	1940	Right parietal region	27	Fibrosarcoma
Abbott and Courville [24]	1942	Fourth ventricle	..	Malignant fibrous meningioma
Abbott and Kernohan	1943	Right temporal, posterior, frontal and anterior occipital regions	14	Fibrosarcoma
Abbott and Kernohan	1943	Left occipital region	21	Fibrosarcoma
Abbott and Kernohan	1943	Left lateral ventricle	4	Fibrosarcoma

* Their cases 24 and 25 may have been instances of malignant meningioma, but it is not possible from their descriptions or photomicrographs to determine the type.

histologically to be of a high grade of malignancy, a fact which was borne out by the short survival period of eight weeks.

All the tumors thus far reported on (table 2) occurred in the cerebral hemispheres; no tumor of this type has originated within the cerebellum except those in 3 cases reported by Foerster and Gagel,[22] in which the tumors arose in the arachnoid over the cerebellum. On the basis of the smaller tumors occurring deep in the brain, in the white centrum, and of the symptoms which they produce early in their development, it might be surmised that most of the tumors originate in the white substance rather than in the cortex, and that in no instance did they originate in the meninges over the surface.

22. Foerster, O., and Gagel, O.: Das umschriebene Arachnoidealsarkom des Kleinhirns, Ztschr. f. d. ges. Neurol. u. Psychiat. **164**:565-580 (Jan. 23) 1939.

Multiple primary tumors have been reported by 2 authors.[23] In each case two separate neoplasms were described: In Houlton's [23b] case one lesion occurred in the genu of the left internal capsule and the other in the subcortex of the left frontal lobe; in Cottrell's [23a] case one was in the left frontal region and the other in the temporal region. In 2 reported cases a tumor occurred in one of the lateral ventricles: In Bailey's [4] case it was in the right lateral ventricle and in 1 of our cases in the left lateral ventricle (table 1). In a case reported by Abbott and Courville [24] the tumor occurred in the fourth ventricle. The latter fact necessitates the differentiation of fibrosarcoma and ventricular meningioma.

The histologic characteristics of fibrosarcoma of the brain are the same as those of fibrosarcoma situated elsewhere: Slender to fat spindle and oval cells occur in a matrix of collagen and reticulin fibrils, and there may be great variation in the cellular pattern and the degree of malignancy. Although the ordinary fibrosarcomatous pattern predominates, "pseudoalveolar," myxomatous and perivascular types have been described, and, in fact, were seen by us in various portions of the 3 fibrosarcomas we nave described. The fact that some tumors apparently are composed almost wholly of one or the other of these cell types had led to their being reported as "reticulum cell sarcoma," "primary reticuloendothelioma," *"sarcome primitif,"* "alveolar sarcoma," "sarcoma magnocellulare" and "fibromyxoma," all of which appear to be forms of fibrosarcoma.

Although the 3 tumors we have described tended to remain fairly localized, the meninges occasionally were seen to be grossly involved. Further scrutiny disclosed evidence of both direct extension and perivascular involvement arising from the primary neoplasm. In 1 of our cases there were distinct leptomeningeal implants over the brain stem and cerebellum; in no instance in our experience has there been a diffuse "infection" of the intracranial or spinal meninges. However, in case 23 in Bailey, Buchanan and Bucy's [25] series, the lesion appears to have been an exception, and a most unusual specimen, for it had spread throughout the subarachnoid spaces from the optic chiasm to the cauda equina but had nowhere involved the nerve tissue. In this case, and in their cases *22* and *26*, the tumors appear to have been unique; it is suggested that they may have originated from connective tissue of the leptomeninges (pia or arachnoid [?]); we know of no other cases in the literature similar to them.

Of utmost importance appears to be the degree of malignancy observed in these fibrosarcomas. A survey of the literature discloses a series of neoplasms characterized histologically by gradual transitional stages from the comparatively benign fibroma to the malignant fibrosarcoma. It seems possible that these tumors may be divided into three types: (1) fibroma,[26] (2) fibroblastoma [27] and (3)

23. (*a*) Cottrell, L.: Primary Fibrosarcoma of the Brain, Arch. Path. **27**:895-901 (May) 1939. (*b*) Houlton, T. L.: Report of a Case of Multiple Sarcoma of the Brain, Nebraska M. J. **11**:169-174 (May) 1926.

24. Abbott, K. H., and Courville, C. B.: Intraventricular Meningiomas: Review of the Literature and Report of Two Cases, Bull. Los Angeles Neurol. Soc. **7**:12-28 (March) 1942.

25. Bailey, P.; Buchanan, D. N., and Bucy, P. C.: Intracranial Tumors of Infancy and Childhood, Chicago, University of Chicago Press, 1939, pp. 90-113.

26. Recently we have seen an excellent example of this type of tumor, a pure fibroma, without arachnoid cap cells. The growth apparently had remained silent in the right island of Reil, the patient having died of other causes.

27. The tumors reported by Mallory (Mallory, T. B.: Primary Fibrosarcoma of Brain: Primary Carcinoma of Pancreas, New England J. Med. **203**:174-177 [July 24] 1930); Meyer and Scheller (Meyer, H. H., and Scheller, H.: Ueber ein Fibromyxom des Gehirns, Virchows

(Footnote continued on next page)

fibrosarcoma. The first type is composed of mature connective tissue, characterized by adult, slender spindle cells, with a matrix of collagen and reticulin fibrils and with no mitotic figures. Grossly, the tumor is a hard, firm mass, or at times a soft mass, and thus far has been reported as occurring only in the ventricles (fibroma, fibrous meningioma, and the like). The second type (benign fibroblastoma) is here considered to represent those tumors which show evidence of growth or expansion and cellular activity. It consists of fat or oval spindle cells, which stain darker with hematoxylin and contain a little more cytoplasm than do the cells of the fibroma. The third, and malignant, type is the fibrosarcoma. It is recognized that the fine point of distinction between fibroblastoma and fibrosarcoma is not always easily made. Nevertheless, it seems to us that when mitoses are seen as frequently as once in every two "low power fields" and when the darker-staining nuclei are surrounded by abundant cytoplasm, a tumor so characterized should be considered a fibrosarcoma.[28]

In differentiation of this malignant connective tissue tumor from gliomas, the glioblastoma containing fibrous tissue probably is more easily confused than any other form. But the cellular pleomorphism of the delicate glial cells, among which typical glioblasts, astroblasts and astrocytes with glial fibers may be identified, should enable one to distinguish between the two tumors.

Mention must be made here of the malignant "stromal" meningioma, which differs from fibrosarcoma only in the fact that it sometimes has the characteristics of meningioma, that is, whorls and arachnoid cap cells appearing in various forms (benign to malignant). But these characteristics may be lacking, so that the malignant "stromal" meningioma may be histologically identical with the fibrosarcoma. This observation coincides with the results of the investigation by Turner, Craig and Kernohan,[29] who, in reviewing the malignant meningiomas, stated: "One type appeared to resemble typical fibrosarcoma with streaming cell bundles and a definite tendency toward an interlacing pattern." Hence, some of the tumors appearing within the brain obviously are identical with those occurring on the surface. This is not surprising when the meningiomas are studied on the basis of their histologic structure, the method of approach used by O. T. Bailey,[30] Foot,[31] one of us (Kernohan [32]) and Courville and Abbott,[33] who showed that the

---

Arch. f. path. Anat. **300**:473-486 [Aug. 6] 1937); Alpers, Yaskin and Grant (Alpers, B. J.; Yaskin, J. C., and Grant, F. C.: Primary Fibroblastoma of the Brain, Arch. Neurol. & Psychiat. **27**:270-281 [Feb.] 1932); Baker and Adams (Baker, A. B., and Adams, J. M.: Primary Fibroblastoma of the Brain: Report of a Case, Am. J. Path. **13**:129-138 [Jan.] 1937); Ferens (?) (Ferens, E.: Case of Primary Reticulo-Endothelioma of Central Nervous System, Neurol. polska **21**:387-402, 1938), and possibly others, seem to be representative of these two types.

28. Among the tumors reported in the literature, those of Houlton [23b]; Zagni (Zagni, L.: Sur deux cas de sarcome primitif des hémisphères cérébraux: contribution anatomo-pathologique et histogénétique, Néoplasmes **5**:159-173, 1926); Bailey [4]; Foot and Cohen [44]; Armenise (Armenise, P.: Un caso di sarcoma cerebrale [Note cliniche ed istopatologiche], Cervello **11**:25-39 [Jan. 15] 1832); Balduzzi [45]; Hsü,[7] and Cottrell [23a] appear to be typical fibrosarcomas. The tumors reported by Foerster and Gagel [22] may well have been malignant meningiomas.

29. Turner, O. A.; Craig, W. M., and Kernohan, J. W.: Malignant Meningiomas: A Clinical and Pathologic Study, Surgery **11**:81-100 (Jan.) 1942.

30. Bailey, O. T.: Histologic Sequences in the Meningioma, with a Consideration of the Nature of Hyperostosis Cranii, Arch. Path. **30**:42-69 (July) 1940.

31. Foot, N. C.: Meningioma, Arch. Path. **30**:198-211 (July) 1940.

32. Kernohan, J. W.: Tumors of the Spinal Cord, Arch. Path. **32**:843-883 (Nov.) 1941.

33. Abbott and Courville.[24] Courville, C. B., and Abbott, K. H.: The Angioblastic Group of Meningiomas: A Study of Thirteen Verified Cases, Bull. Los Angeles Neurol. Soc. **5**:47-72 (April) 1940.

meningioma consists of two cell types, first, the arachnoid covering cap cell, which is assumed to represent the adult, or differentiated, tumor cell and, second, stromal tissue, consisting of collagenous connective tissue. It has been shown by Abbott and Courville [24] that the so-called meningiomas situated in the ventricles all are of fibrous or fibroblastic nature, that they lack the arachnoid cap cell element and that, by definition, they arise from connective tissue. They are of the same histologic type as the fibrosarcoma, differing only in the degree of cellular differentiation they possess. This statement, however, does not take into consideration their origin, nor does it prove or disprove their origin from the so-called perithelial cells or from other connective tissue elements of the blood vessels; it merely tends to substantiate the assumption that both tumors can arise from any connective tissue situated in or on the surface of the brain.

PERIVASCULAR SARCOMA

We studied 7 tumors which we classified as perivascular (perithelial) sarcoma. Five of these tumors were obtained at necropsy, and 2 were specimens of tumor tissue removed for biopsy. In the cases of the latter an attempt had been made to extirpate the malignant neoplasm. Because of the infrequency with which tumors of this type have been reported, it seems worth while to report in some detail 3 of the 7 cases in this group. In case 4 (table 1) the lesion was typical of the localized tumor mass not uncommonly encountered in the cerebellum, and in case 8 (table 1) we found that this type of tumor may also occur in the cerebrum. In cases 5 and 6 (table 1) the lesions represented rarer forms, being the only ones of their kind in our experience. Moreover, the literature does not contain any similar cases, except for a case reported by Fried,[21a] in which the lesion seems to have been much like the lesion in our case 6.

CASE 4.—*Perivascular sarcoma of cerebellum.*

A boy aged 17 years (case 4, table 1) entered the Mayo Clinic on Jan. 12, 1939, complaining of headache, vertigo and tinnitus of six weeks' duration. Up to three weeks prior to his admission the headache had been intermittent; it then had become constant and was associated with nausea and vomiting. During the previous two weeks diplopia and unsteadiness of gait were in evidence. Examination disclosed that the boy was acutely ill; he presented acute bilateral papilledema with retinal hemorrhages, nystagmus, a cerebellar type of incoordination of the right extremities and moderate stiffness of the neck. It was evident that a lesion was situated in the posterior cranial fossa and that this lesion probably was a neoplasm. Since the patient's condition was critical, it was thought best to relieve the internal hydrocephalus by ventriculostomy, which was carried out on January 18. Intracranial pressure was found to be greatly increased. The patient responded well immediately after the procedure, but he died suddenly thirteen hours later.

Complete necropsy, performed soon after the death of the patient, revealed a severe grade of increased intracranial pressure, with acutely herniated cerebellar tonsils and hydrocephalus. Of more importance was the enlargement of the right cerebellar hemisphere, which on section was observed to contain, beneath its surface, a multinodular neoplasm. The nodules, connected by bands of tumor tissue, were each 1 to 2 cm. in diameter. The neoplasm was easily separated from the cerebellum by finger dissection, for the brain tissue around it was obviously degenerated, so that it had a pseudoencapsulated appearance. Section of the cerebrum disclosed only a slight degree of edema and hydrocephalus.

Microscopically (fig. 4*a*), the tumor was composed of small, irregular round or oval cells, with rather deeply staining, round to oval nuclei. Moderate cellular pleomorphism was evident in places, but for the most part the cells were surprisingly uniform in size. Their nuclei

contained only moderately coarse chromatin, with one to three nucleoli. Mitotic figures were encountered frequently. The cytoplasm varied in amount; it was nowhere abundant and generally was rather indistinct in outline. The cells obviously were of a coarser structure than those of glial origin. Glial fibers were absent. Most striking was the distinctly perivascular arrangement of the cells; however, within the tumor mass, alinement of the cells in columns and strands also was noted. Sections from the edges of the tumor disclosed that the growth had invaded the brain tissue by perivascular extension, but of more importance and of greater prominence was the evidence of its propagation by way of the walls of the vessels as it advanced. Tumor cells in many of these portions appeared to be "budding" out of the walls of small blood vessels. This, of course, is only an interpretation, and the opposite view, that of invasion of the walls of the vessels by tumor cells, could be advanced as easily as ours. The reticulin pattern, however, tends to substantiate the first conclusion.

In the sections stained according to the Perdrau and Laidlaw technics, reticulin was present among the cells and was intimately associated with them, particularly when the cells were

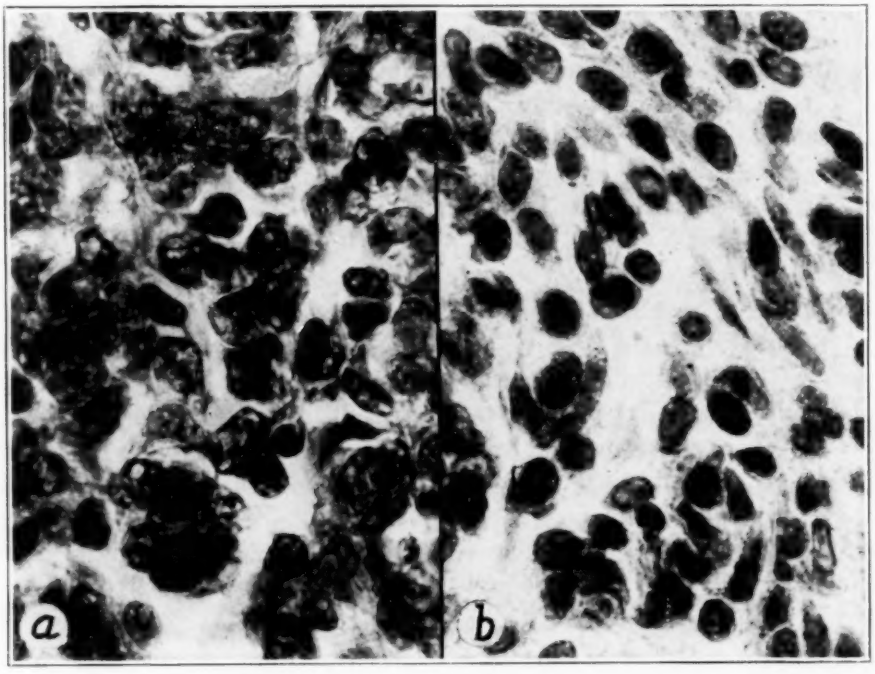


Fig. 4.—(*a*) Perivascular sarcoma in case 4: Irregular, oval nuclei, with many mitoses, are seen in this section; the tumor itself seemed to be highly malignant. (*b*) Perivascular sarcoma in case 7: Oval and spindle-shaped nuclei, with mitotic figures, are here evident. Hematoxylin and eosin; × 900.

viewed in their perivascular arrangements. In many areas rings of reticulin about the vessels (cut in cross section) were presented, whereas in other areas reticulin appeared to radiate from the vessels (broken ring arrangement, fig. 4 *b*). In the lesion in case 7 (fig. 5 *a* and *b*) and in the lesion in case 9 (fig. 6) a similar arrangement was seen. When vessels appeared in longitudinal section, strands of reticulin were seen along both sides and on the vessels. There were no tumor cells in the meninges, except in a small portion deep in the cerebellar folia.

The blood vessels in the surrounding cerebellar tissue seemed to be more numerous than normal. The intervening nerve tissue was in a state of degeneration, and zones of necrosis were present.

The diagnosis was perivascular sarcoma of the cerebellum.

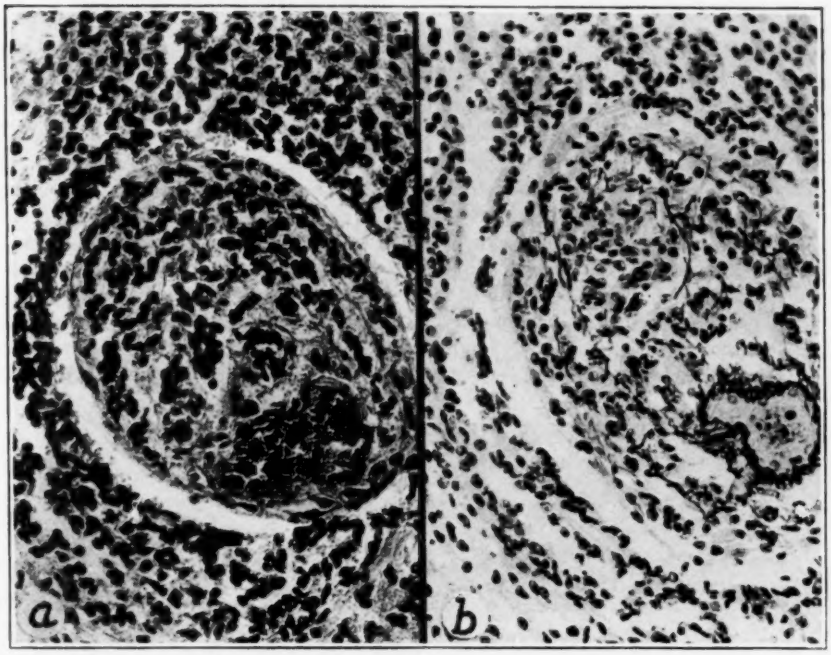


Fig. 5 (case 7).—Perivascular sarcoma: (*a*) Tumor cells here seemed to be "budded off" from the wall of a vessel (hematoxylin and eosin; × 190); (*b*) silver impregnation of the same vessel sectioned a few microns away from the level seen in *a*, showing delicate reticulin rings (Perdrau technic; × 190).

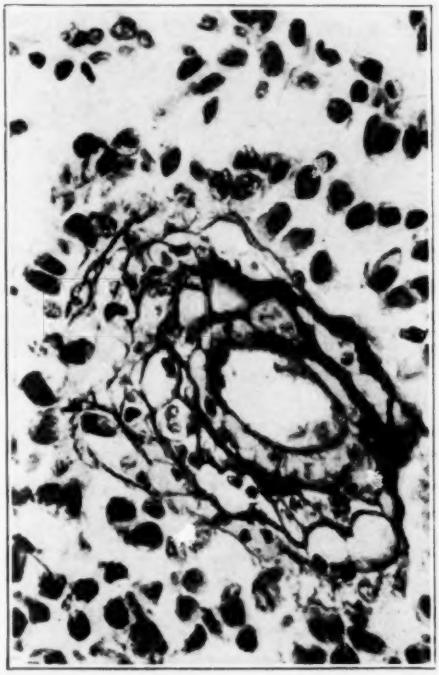


Fig. 6 (case 9).—Perivascular sarcoma: Heavy reticulin rings appear in this section (Perdrau technic; × 415).

CASE 5.—*Multiple primary perivascular sarcomas of the right lateral and the fourth ventricle.*

A male physician aged 57 (case 5, table 1) visited the clinic because of a suspected abdominal mass. During the two weeks previous to his admission he had had an attack of influenza, followed by nausea, occasional vomiting and one short episode of vertigo. While he was undergoing examination, blurred vision and nystagmus with dizziness and vomiting developed.

General examination did not reveal an abdominal mass, but neurologic investigation disclosed weakness of the left rectus lateralis muscle, with nystagmus (more pronounced in the right eye than in the left), incoordination of all extremities, inattention and somnolence. Papilledema of the optic disks was not present, but the veins were engorged. Spinal tap in the lumbar region yielded cerebrospinal fluid, of which the total protein content was 300 mg. per hundred cubic centimeters. There were 104 cells per cubic millimeter, all of which were lymphocytes. The electroencephalogram was recorded as disclosing localization of delta waves in the right temporal region, of grade 3 plus intensity. The condition of the patient steadily failed, and he became more confused and disoriented. Hemiparesis developed on the left, and

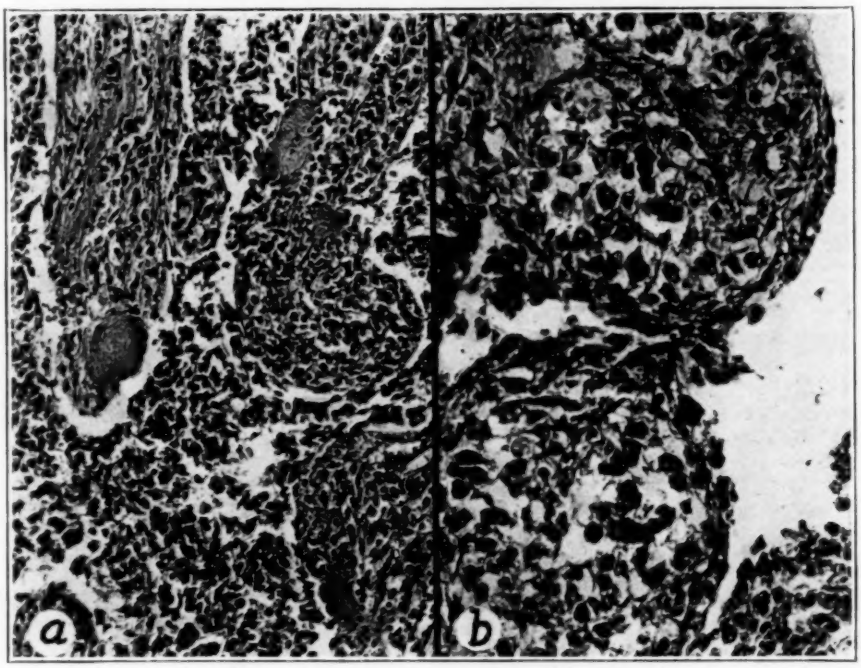


Fig. 7 (case 5).—Perivascular sarcoma: (*a*) The tumor is arising in (or invading) the choroidal tufts of the lateral ventricle (hematoxylin and eosin; × 150); (*b*) the choroidal tufts are here seen to be distended with sarcoma cells, and flattened ependymal cells also are present (hematoxylin and eosin; × 280).

coma occurred. The patient remained in this state for four weeks, at the end of which he died. Therefore he lived only six weeks from the time of onset of the symptoms.

Necropsy, limited to the head, was performed in the patient's home city, and the brain was sent to the clinic for study. Sections of the specimen disclosed multiple primary tumors; one was situated in the right lateral ventricle, and another filled the fourth ventricle. The tumor in the right cerebral hemisphere filled and distended the parietal and occipital portions of the lateral ventricle and appeared to be invading the nerve tissue of these lobes and to occupy the choroid plexus completely. The mass was approximately 5 cm. in diameter. The cut surface was pinkish white and of moderately soft consistency. A tumor of similar description occupied the fourth ventricle and invaded the superior part of the pons Varolii and the cerebellar vermis; it was 4.5 cm. in diameter. It, likewise, appeared to be invading the adjacent nerve tissue. Blocks of tissue were taken from the tumor masses, the walls of all the ventricles and the cerebral and cerebellar hemispheres adjacent to and at a distance

from the lesions. These blocks were sectioned and stained with hematoxylin and eosin and Mallory's phosphotungstic acid, and by the Mallory-Heidenhain, Perdrau and eosin, Orlandi, Bodian and cresyl violet methods.

Histologic study revealed these neoplasms to be rather typical perivascular sarcomas, which differed from the others studied in this series in that they were of a somewhat higher degree of malignancy, with considerably more cytoplasm around the small, hyperchromatic nuclei and with many more mitoses. The two tumors involved the choroid plexuses in a most interesting manner. Practically every tuft in both plexuses was distended with tumor cells, but each more or less retained its normal covering of ependymal cells (fig. 7 *a* and *b*). There was invasion of the surrounding nerve tissue for a short distance (3 to 4.5 cm.), the extension being primarily of a perivascular nature. The process subsequently had broken through the Virchow-Robin boundaries and had then spread diffusely, with resultant degeneration of the nerve tissue. However, the invasive tendency was much greater than that seen in the other tumors, which occurred in the form of a more localized mass. The neoplasm in the lateral ventricle had grossly invaded the corpus callosum, the thalamus, the optic radiation, the hippocampus and associated structures, but did not involve the walls of the third ventricle; neither was there metastatic "infection" of the walls of this ventricle. The neoplasm in the fourth ventricle had invaded the medulla to various depths, from one-third to two-thirds the distance ventrally and caudally, to just below the lower border of the vermis. Tumor tissue, also, was present in the inferior third of the cerebellar hemisphere (including the vermis), having grown to the surface in this situation, with minor involvement of the leptomeninges. A section made through the region of the foramen of Magendie disclosed a small cord of tumor extending from the intraventricular mass into the subarachnoid space and out over the vermis. In many areas of the vermis and the lower medial and inferior portions of the cerebellar hemispheres there was partial to almost complete disorganization of the granular and molecular layers, due to infiltration by tumor cells and the subsequent degeneration of nerve tissue.

There seemed to be no doubt that two primary ventricular tumors had arisen in this case and that both were of approximately the same size and of the same grade of malignancy, although the tumor of the fourth ventricle was perhaps slightly more anaplastic, with more invasive tendencies. Although it was not possible to prove the origin of these tumors beyond a doubt, we believe they most probably arose from the connective tissue elements of the choroid plexus, or possibly from the vascular adventitial cells of the pia mater, which makes up the velum of the superior tela.

The diagnosis was multiple perivascular sarcomas.

CASE 6.—*Diffuse perivascular sarcomatosis of the central nervous system.*

A man aged 41 (case 6, table 1) consulted the clinic because he had experienced periods of depression and severe pains in the head. Investigation of his past social and business experiences seemed to provide adequate reason for the depression, but not for the pains in the head. While the patient was under observation, the pains became more intense, and he displayed periods of severe psychotic disturbance, which appeared to be of organic type. The neurologic signs varied from day to day, but signs of bilateral involvement of the pyramidal tract and nystagmus were fairly constant. Although the signs and symptoms indicated a diffuse process, there was evidence that a lesion was present in the frontotemporal region; this was corroborated by electroencephalographic tracings. The patient died in a state of coma eleven weeks after the onset of symptoms.

Careful study of the gross specimen obtained during a complete necropsy revealed a large, degenerating tumor situated in the right frontotemporoparietal region. Sections, taken from nearly every portion of the brain and spinal cord, disclosed diffuse, widespread perivascular sarcomatosis. Practically no part of the brain was unaffected by the neoplasm, for tumor cells were present around the majority of the vessels in every section, including those of the spinal cord (fig. 8 *a*). Although the tumor tissue was situated predominantly in the cerebral hemispheres, tumor cells could be seen easily around tiny capillaries at various levels in the spinal cord. Of further interest was the fact that the cortex in the cerebral hemispheres had been relatively spared, whereas the white matter was densely infiltrated. To be sure, this was not altogether the situation in every field; yet on the whole there was definitely less cortical infiltration with perivascular tumor cells.

That the sarcoma cells were not disseminated throughout the central nervous system by the cerebrospinal fluid was evidenced by the slight leptomeningeal invasion of the tumor, by the apparent simultaneous origin of the two tumors and by the widespread, diffuse character of the growth (fig. 8 *a*). Tumor cells, also, were seen growing out of the walls of the smallest capillaries, a fact which precludes the possibility of the origin of the tumor from

the pia mater, as suggested in another instance by Bailey,[4] for the pial sheaths (Schaltenbrand and Bailey[11b]) do not extend as far as the capillaries. Thus, more evidence in favor of the theory of the origin of perivascular sarcoma from vascular connective tissue was adduced.

The diagnosis was diffuse perivascular sarcomatosis of the entire central nervous system.

In the more recent literature, in the majority of cases in which microscopic identification of the tumor was sufficiently certain to make tabulation of the verified cases reasonably accurate, it was possible to note the sex and the age of the patient and the situation of the tumor. It was surprising to find that in the reported cases nearly all the patients were males, an observation which was true of all the 7 patients in our series who had perivascular sarcoma of the brain (table 1). This unusual preponderance of males very likely has some significance, although it probably would be presumptuous of us to attempt to state what it is. Although the victims of this neoplasm reported on in the literature have represented all

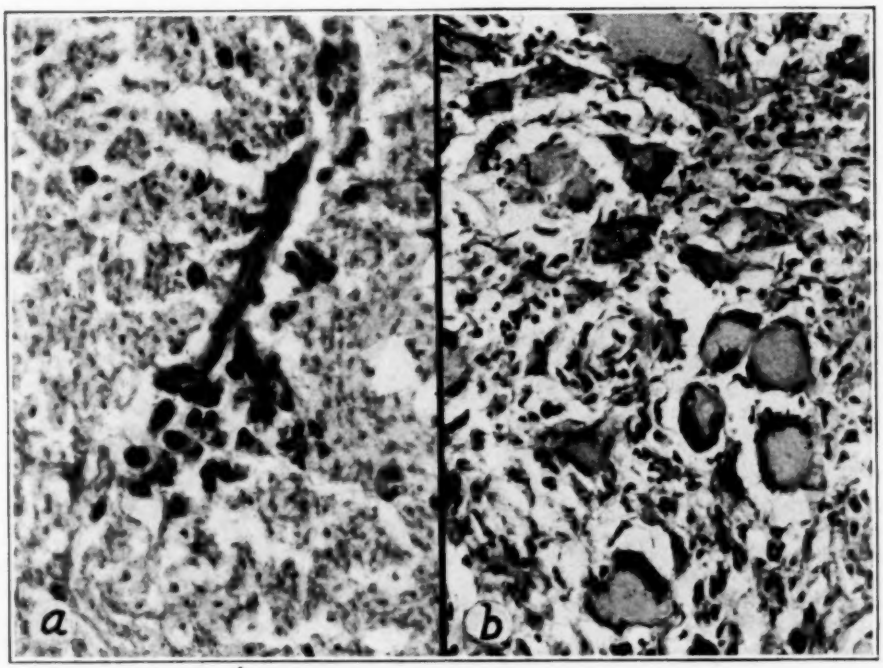


Fig. 8.—(*a*) Small perivascular sarcoma cells (hematoxylin and eosin; × 350) encountered in case 6 are here seen arising from a capillary in the thoracic level of the spinal cord (diffuse perivascular sarcomatosis). (*b*) A giant cell fibrosarcoma of the brain in case 11 (hematoxylin and eosin; × 200).

age groups, from the first to the seventh decade, the lesion most commonly occurred among persons from 30 to 50 years of age. Rarely did the lesion afflict patients in the first decade of life (2 cases), a fact which may be of significance in distinguishing perivascular sarcoma from certain types of glioma (medulloblastoma).

The duration of symptoms in our 7 cases ranged from five weeks to five months. The average duration was two months, a surprisingly short period, particularly when it is compared with the duration of symptoms in the cases of fibrosarcoma (table 1). This short period of survival in our 7 cases corresponds with the period in similar cases reported by other authors and tends to corroborate the histologic evidence of the high grade of malignancy of the perivascular sarcoma.

The postoperative period of survival was known in 5 of our 7 cases, and the approximate time of survival could be estimated in the seventh case. In 1 case death occurred in thirteen hours, in another two days and in a third three days after surgical intervention (table 1). In 2 cases in which operation was not performed the patients died six and eleven weeks respectively after the onset of symptoms definitely referable to the neoplasm. In the final 2 cases in which an operation was done the period of survival was only four and a half and five months respectively (in 1 case the exact date of death after surgical intervention was not obtained).

In 2 of the 7 cases in this group the malignant process was situated in the left cerebellar hemisphere, and in a third, in the right cerebellar hemisphere. In 1 case the tumor was in the right frontotemporal lobe, with diffuse, generalized perivascular sarcomatosis of the central nervous system; in another, in the right temporal lobe, and in a third, in the left frontoparietal region of the cerebral hemispheres. In another case one neoplasm was in the right lateral ventricle and one in the fourth ventricle; however, in summarizing the cases reported in the literature, we found that perivascular sarcoma has no site of predilection and that in various cases all parts of the brain have been involved by it.

Macroscopic inspection, as described in the reported cases and carried out in our 7 cases, revealed that the gross characteristics of perivascular sarcoma range from pseudoencapsulation, as seen in cases 4 and 8, to a diffuse, extensive neoplastic process involving the major portion of one or both hemispheres. In 1 instance [21a] there was only scant gross evidence of what was disclosed microscopically as generalized perivascular sarcomatosis. The tumor in our case 6 undoubtedly was analogous to the one in Fried's [21a] case. Although there was a large mass in case 6, we did not suspect the widespread, diffuse presence of tumor which was disclosed by the histologic sections taken from every part of the brain and spinal cord.

It has been reported, however, that the perivascular sarcoma frequently appears as a fleshy, somewhat firm, localized mass or as a reddish portion of a soft, jelly-like consistency, spreading out as streaks and bands into the surrounding nerve tissue.[34] As far as we are able to determine, our case 5 is the first reported instance of a perivascular sarcoma within the ventricles, although as has been pointed out, the occurrence of intraventricular fibrosarcoma is now well recognized.

The histologic aspects of this tumor may be summarized as follows: There is conspicuous cellularity, with relatively small round or oval, occasionally somewhat spindle-shaped, cells with hyperchromatic nuclei, containing finely granular to slightly coarse chromatin bundles, and a scant to moderate amount of finely granular cytoplasm, which stains much lighter than the nucleus; at times the boundaries of the cells are difficult to outline. We noted only a moderate degree of pleomorphism, but mitoses were frequently encountered. Tiny blood vessels were numerous and were scattered in varied interconnecting patterns throughout the tumor. Reticulin was noted in intimate association with the neoplastic cells in sections prepared according to the Perdrau and the Laidlaw silver impregnation technic. These two technics were of the utmost value in a study of the perivascular nature of the tumors, as is evident in all the more recent reports. The perivascular multiple rings of reticulin mentioned by several authors were seen in all our tumors.

34. Scheinker, I.: Ueber eine seltene zerebrale Tumorart (diffuses perivasculäres Sarkom), mit besonderer Lokalisation im Stirnhirn. Zugleich ein Beitrag zur Frage der sogenannten "Peritheliome" des Zentralnervensystems, Jahrb. f. Psychiat. u. Neurol. **53**:155-163, 1936. Környey.[5] Mage and Scherer.[6] Hassin.[21b]

Sections made through the edges of the tumor and in surrounding nerve tissue served best to reveal the spread of tumor cells through the Virchow-Robin spaces, and not infrequently tumor cells could be seen in intimate contact with, and apparently budding off, the adventitia of the blood vessels. Study of sections prepared according to the Perdrau and Laidlaw technics confirmed this appearance, for the reticulin was noted in "budding rings." The same phenomenon has been reported by other observers.[35] Fried [21a] reported the appearance of mitotic figures in the walls of certain vessels, an observation we were able to confirm. On numerous occasions the tumor cells were seen to be growing apparently from the wall of the vessel both into the Virchow-Robin space and into the lumen of the vessel.

Fried [21a] and Mage and Scherer,[6] among others, emphasized the resistance of the pia-glia membrane to the spread of connective tissue elements in and around the vessels and into the nerve tissue. Study of the tumors in our cases and in other cases has disclosed that apparently after sufficient proliferation in the Virchow-Robin spaces, the tumor cells break through this boundary and diffuse into the surrounding nerve tissue. From the study of the lesion in case 6 and of lesions in other cases we also gained the impression that widespread transformation of normal, benign vascular connective tissue cells into malignant cells occurs.

Review of earlier reports [36] of the so-called perithelioma, variously called "hemangioperithelioma," "periendothelioma," "angiosarcoma," "pial cancer" and *"myxosarcome téléangiectodes,"* has revealed a group of heterogeneous tumors, the identity of which is not at all certain. Prior to present day histopathology, the term "perithelioma" came into common use and was applied to tumors of nearly every organ. These tumors, however, have in recent years been shown to be various different neoplasms. Thus, this once supposedly common tumor, perithelioma, has disappeared. Our careful review of the literature concerned with the so-called intracranial perithelioma has led us to believe that in this instance, also, there is no such tumor.[37] After the tumors which were misdiagnosed as "peritheliomas" are discarded, what remains, we believe, are sarcoma of varying characteristics.[38] On the basis of these varying characteristics, it has been possible

35. Bailey.[4] Környey.[5] Mage and Scherer.[6] Hsü.[7] Fried.[21a]

36. Borst, M.: Die Lehre von den Geschwülsten mit einem mikroskopischen Atlas, Wiesbaden, J. F. Bergmann, 1902, vol. 1, pp. 340-359 and 369-372.

37. Among the tumors accepted by other authors as peritheliomas might be mentioned one reported by Chambard (Carcinome épithélioïde primitif de la pie-mère; lésions consécutives des parois crâniennes; ramollissement cérébral, France méd. **28**:146 and 158, 1881), which appears to be an excellent example of a large meningioma of the sphenoid wing. The lesion in Cornil's case, referred to by Bailey,[4] falls short of possessing the characteristics of a perithelial sarcoma; similar comments might be made concerning other accepted cases of perithelioma. In Cushing's (Notes on a Series of Intracranial Tumors and Conditions Simulating Them: Tumor Suspects, Tumors Unverified, Tumors Verified, Arch. Neurol. & Psychiat. **10**:605-668 [Dec.] 1923) 2 cases of perivascular endothelioma, a tumor which he considered synonymous with perithelioma, in the light of the descriptions of Lindau and Bailey and Cushing,[3] the tumors may have been hemangioblastomas. In his case 20 the lesion may have been a cerebral noncystic hemangioblastoma, and in case 27, a cystic cerebellar hemangioblastoma. Wilson (Neurology, Baltimore, Williams & Wilkins Company, 1940, vol. 2, p. 1217) referred to the tumors in these 2 cases of Cushing and the growth in a case recorded by Spiller as "hemangioperithelioma" and stated that he had seen 1 such tumor; judging from the histories, descriptions and meager photomicrographic evidence presented in Cushing's and Spiller's cases, we consider that the tumor in Wilson's case likewise belonged to the hemangioblastomas. These cases, among many others, fail to present evidence in support of the existence of such a tumor as the perithelioma.

38. As early as 1906, Carless (Some Recent Reports Concerning Endotheliomata and Peritheliomata, Practitioner **76**:228-242 [Feb.] 1906) had begun to form the same conclusion.

to divide perivascular sarcomas into several fairly well defined types. For the sake of completeness, in spite of our doubts as to its identity, the so-called perithelioma will be included herein.

1. *Perithelioma.*[39]—This tumor is a local mass with apparent or actual encapsulation occurring in the meninges (dura and pia mater) and within the nerve tissue, as described by numerous authors,[40] such tumors are here considered to form a heterogeneous group.

2. *Perivascular (Perithelial) Sarcoma* (group A).—Cases of this lesion were present in our series (cases 4, 7 and 8); the tumor has been reported elsewhere as occurring in the form of one or more local masses [41] within the brain (any part), surrounded by a pseudocapsule of degenerating brain tissue and with obvious, but limited, perivascular extension of tumor cells into the surrounding nerve tissue, in which evidence of its perivascular origin frequently was prominent. Meningeal extension of the tumor was minor and was confined to the adjacent leptomeninges.

3. *Diffuse Sarcomatosis* (group B, type 1.)—Fried's [21a] case of diffuse perivascular sarcomatosis and our case 6 are the only representatives of this type, although less extensive diffusion occurred in many of the other tumors. In Fried's [21a] case small round sarcoma cells were present around the blood vessels through a large part of the brain, with minimal extension into the meninges, but with only one small zone of invasion of the nerve tissue. In our case, in addition to the perivascular sarcomatosis in the cerebrum and cerebellum, the process consisted of definite involvement of the spinal cord.

---

39. The tumor in the case of Achmatowicz and Borysowicz (Perithelioma of the Dura Mater, Polska gaz. lek. **12**:757-758 [Sept. 24] 1933) was reported as a perithelioma of the dura mater occurring after trauma to the skull and osteomyelitis (?), but the authors failed to describe the microscopic appearance; neither were there photomicrographs. Consequently, their tumor cannot be accepted as one of this doubtful group.

40. Arndt, R.: Ein cancroid der Pia mater, Virchows Arch. f. path. Anat. **51**:495-506 (Dec. 17) 1870. Arnold, J.: Ein Myxosarcoma teleangiectodes cysticum der Pia mater der linken Grosshirnhemisphäre, ibid. **51**:441-457 (Dec. 17) 1870. Besold, G.: Ueber zwei Fälle von Gehirntumor (Hämangiosarkom oder sogenanntes Peritheliom in der Gegend des Dritten Ventrikels) bei zwei Geschwistern, Deutsche Ztschr. f. Nervenh. **8**:49-74 (Dec. 30) 1895. Eberth, C. J.: Zur Entwickelung des Epithelioms (Cholesteatoms) der Pia und der Lunge, Virchows Arch. f. path. Anat. **49**:51-63 (Dec. 11) 1870. Hart, C.: Ein Endothelioma perivasculare (Perithelioma) piae matris mit sekundärer Cystenbildung im Gehirn, Ztschr. f. Krebsforsch. **11**:283-293, 1912. Janssen, V.: Ein Sarcom der Pia mater, Virchows Arch. f. path. Anat. **139**:213-232 (Feb. 5) 1895. Lissauer, M.: Ein Peritheliom der Pia mater spinalis, Centralbl. f. allg. Path. u. path. Anat. **22**:49-52 (Jan. 31) 1911. Pianese, G.: Peritelioma durae cerebri, Gazz. internaz. di med. **6**:229-240, 1903. Waldeyer: Die Entwickelung der Carcinome, Virchows Arch. f. path. Anat. **55**:67-159 (June 13) 1872. Wätzold, H.: Ein Peritheliom des Plexus choroideus des linken Seitenventrikels, Beitr. z. path. Anat. u. z. allg. Path. **38**:388-394, 1905. Brower, D. R., and Wells, H. G.: A Periendothelioma of the Dura Mater Involving the Cranial Nerves, Am. J. M. Sc. **122**:32-40 (July) 1901.

41. Undoubtedly, some tumors fall midway between group A and group B, and others, the identity of which, for various reasons, cannot be established with certainty, have not been classified. This is the situation with respect to the tumors described by Haeger (Ausgebreitetes Endotheliom der inneren Meningen des Gehirns, Monatschr. f. Psychiat. u. Neurol. **30**:86-99, 1911), Hashimoto (Zur Kenntnis der Zylindrome und Peritheliome des Gehirns, Arb. a. d. Neurol. Inst. a. d. Wien. Univ. **29**:357-367 [Sept. 1] 1927), Greenfield (The Pathological Examination of Forty Intracranial Neoplasms, Brain **42**:29-85 [April] 1919), Coste and Levy (Ein Fall von Peritheliom der Gehirns, Arch. f. klin. Chir. **96**:1049-1068 [Dec. 21] 1911), among others, whereas Bailey,[4] Hsü,[7] Stevenson and Hyslop (Perithelioma of the Brain: A Case of Multiple Primary Peritheliomata; Discussion of Histopathology, M. Clin. North America **14**:451-461 [Sept.] 1930), Bailey and Ley (Estudio anatoma-clínico de un caso de ocurencia simultánea de dos tumores [glioma y sarcoma] en el hemisferio cerebral de un niño, Arch. de neurobiol. **14**:673-690, 1934), Bailey and associates [25] (case 22) and others have described excellent examples of perivascular sarcoma.

4. *Diffuse Perivascular Sarcoma* (group B, type 2).—Large portions of nerve tissue are involved. We have seen several such tumors, and a number of cases have been reported.[42] In such cases the leptomeninges, as is true of them in the presence of tumors of group A and tumors of group B, type 1, were involved only to a minor degree, in spite of the widespread presence of "perivascular" sarcoma cells in the nerve tissue and around the blood vessels. In nearly every case the white centrum, the corpus callosum and the basal ganglia were the structures involved, so that there was a tendency for the cortex to be spared. Because of the invasive nature and other evidences of extremely high malignancy, the two intraventricular tumors we have described in case 5 might properly be classified in this group (group B, type 2), rather than in an additional subgroup. To place them in a subgroup would unduly complicate an otherwise simple classification.

5. *True Diffuse Leptomeningeal Sarcomatosis* (group C).—We have no cases of this lesion to report. Suffice it to say that we consider the tumor to be a widespread malignant transformation of the pia mater, whereas a similar process occurring in the arachnoid is the most probable cause of the diffuse meningiomatosis.[43]

SARCOMA OF UNKNOWN TYPE

In 2 cases in our series the tumor was not typical of either fibrosarcoma or perivascular sarcoma. It appears, however, that the lesion in the first case was a type of fibrosarcoma and that the tumor in the other case may have been a perivascular sarcoma.

CASE 11.—*Probable primary malignant giant cell fibrosarcoma.*

A woman aged 34 (case 11, table 1) came to the clinic moribund. She had complained of frequent frontal headaches, vomiting, scotomas and diplopia and had had jacksonian and grand mal seizures in the preceding nine months. She died a few hours after her admission to the hospital.

Postmortem observations, except for an intracranial tumor, were not remarkable. In the brain we observed a soft, reddish mass in the tip of the left temporal lobe, and coronal sections disclosed that the tumor had extended around the sylvian fissure and into the left frontal lobe. Histologic study of sections of the tumor revealed that it was composed of innumerable large and small multinucleated giant cells (fig. 8 *b*), dispersed in a stroma of which the general architecture varied from very loose to compact spindle cells, containing variable amounts of cytoplasm, and in which collagen and reticulin were seen. Some of the nuclei in the stroma appeared to have been extruded from the large giant cells; others, which had a small amount of cytoplasm similar to that of the giant cells, may well have been small giant cells. The giant cells showed wide variation in size, shape and structure; they ranged in size from 7 to 90 microns. Their nuclei stained darkly with hematoxylin and eosin and contained coarse groups or clumps of chromatin; these clumps collected at the periphery to form a dark rim, which lent a distinct and clear outline to the nucleus. One to three nucleoli occasionally were identified in the nucleus. Not infrequently lighter-staining nuclei were present. The size of the cell had no relation to the number of nuclei present in them, for small cells with six to eight nuclei were packed in so closely that the intervening cytoplasm appeared to be scanty. Large cells which had from one or two to as many as twenty-four nuclei were commonly encountered. The nuclei varied from 5 or 7 to as much as 40 microns in diameter. Occasionally giant nuclei, almost completely filling the cell, appeared in chains or in U and S shapes, possibly the result of fusion of several small nuclei. Swelling, with disintegration of these giant nuclei, was occasionally seen. Thus there were three morphologic types of nuclei: (1) small, irregularly oval nuclei; (2) large, lobulated nuclei, and (3) small to medium-sized "turbinated" nuclei with processes attached to their apexes. The positions of the nuclei varied: Some were clumped together in the center or at the periphery of the cell in a spherical manner; some were clumped in disconnected chains at the periphery; some were located only

42. Bailey.[4] Környey.[5] Mage and Scherer.[6] Hassin.[21b] Scheinker.[34]

43. Brown, M. H., and Kernohan, J. W.: Diffuse Meningiomatosis, Arch. Path. **32**:651-658 (Oct.) 1941.

at one end; some were scattered around the periphery, and some were scattered over the cell in haphazard manner. They were usually round or oval, but occasionally were bean shaped. Mitotic figures of all sizes and shapes frequently were seen in them.

The cytoplasm in the sections treated with hematoxylin and eosin stained a bright pinkish red and had a fairly homogeneously colored ground glass appearance. The shape and amount varied, from a thin rim around a nucleus to large sheets, but nearly all cells in the paraffin sections had a crude, irregular stellate outline, the points of which seemed continuous with collagenous and reticulin fibrils. When the nuclei were studied in frozen sections, they lost this appearance and were more often round to oval, and not infrequently elongated, a difference suggesting that the morphologic characteristics previously described were accentuated by shrinkage in paraffin-fixed sections. The reticulin fibrils were well delineated in sections prepared according to the Perdrau and Laidlaw technics of impregnation. Of particular interest, but of questionable significance, were eosinophilic, globoid or rounded masses which frequently were encountered in the cytoplasm. Their presence was accentuated by special stains, and their eosinophilic, conglomerate, chromatin-like structure suggested that they were degenerated forms. No undisputed evidence of phagocytic activity was detected in any section.

The cells and intercellular fibrils stained deep blue with the Mallory-Heidenhain method and red with Mallory's phosphotungstic acid; thus, their connective tissue nature was demonstrated. In the sections prepared by Mallory's method there was no evidence of neuroglial fibrils or sucker feet. The results of these staining processes confirmed the mesodermal character of the turbinated giant cells with attached processes and distinguished them from glial types and the sections prepared according to the Río-Hortega and Cajal technics and aided in ruling out any glial characteristics.

The blood vessels frequently were thrombosed, apparently because of invasion of the walls and lumens by tumor cells. Frequently, large and small multinucleated cells were attached to the somewhat thickened vascular wall, as if they had arisen directly from the wall, the frequency of this occurrence suggesting that this relation was more than casual. There was, however, little perivascular extension of the tumor into the surrounding portion of the brain; instead, the tumor had directly invaded the surrounding tissue, an action similar to that of primary fibrosarcoma of the brain.

In quest of further distinction of the cells from ganglion cells, we studied sections treated with cresyl violet. Such a study did not show that the cells resembled any type of neuron.

Since by far the majority of the cells were giant cells, the possibility that the lesion might be a rhabdomyosarcoma was thoroughly investigated, but cross striations could not be seen in any of the sections. Multiple small hemorrhagic foci were present throughout the tumor, but they were situated most frequently around the periphery and in the adjacent nerve tissue.

Although we were unable to find any similar tumor in our series or a report of one in the literature, we recognized that tumor giant cells have been reported as occurring in other types of intracranial sarcoma (Foot and Cohen,[44] Balduzzi[45]) and that such cells constitute the characteristic feature of certain fibrosarcomas of bone. Therefore, we believe that the tumor was most probably a primary malignant giant cell fibrosarcoma of the brain.

CASE 12.—*Infiltrating neoplasm of unknown type in the left frontoparietal region.*

A man aged 40 (case 12, table 1) had experienced jacksonian seizures and progressive hemiparesis on the right side, together with motor aphasia and dull headache in the three months previous to his admission. At operation an infiltrating neoplasm of the left frontotemporal region was removed. The patient has survived the operation about three years. When we last had word from him, we learned that there was evidence of a return of the neoplasm and that his health was failing rapidly.

Histologically, the tumor was very cellular, being composed of small, round, oval, indented, bean-shaped, or even lobular, basophilic nuclei, characterized by only moderate pleomorphism (fig. 9 *a* and *b*). Each cell contained a small amount of poorly staining cytoplasm, of indefinite outline. Nucleoli could be clearly demonstrated only in the large nuclei, which were few. The cells were dispersed in a meshwork of collagen, in which intercellular reticulin fibrils were demonstrated by the silver impregnation method of Laidlaw. Numerous small

44. Foot, N. C., and Cohen, S.: Report of a Case of Retotheliosarcoma (Reticulosarcoma) of the Cerebral Hemisphere, Am. J. Path. **9**:123-132 (Jan.) 1933.

45. Balduzzi, O.: Sarcoma magnocellulare cerebrale primitivo (contributo anatomopatologico), Riv. sper. di freniat. **62**:695-705 (Sept. 30) 1938.

capillaries, the walls of which were composed of thin layers of endothelial cells surrounded by a thin rim of fibrous tissue, collagen and tumor cells, permeated the tumor. Frequently several large nuclei in a scanty cytoplasm of indefinite outline were situated around and in the walls of these minute vessels. Small zones of necrosis were a prominent feature. Although the tumor obviously was a sarcoma, various areas when viewed alone gave the impression of being granulation tissue, lymphadenoma (Hodgkin's disease), lymphosarcoma and even uterine endometrial (stromal) sarcoma respectively. As far as we were able to determine, this tumor could not be classed with any of the now fairly well defined and recognized types of intracranial sarcoma. It likewise differed from the many bizarre forms of dural sarcoma, one of which may possibly have been its origin. It seems more likely, however, that the tumor originated from the leptomeninges on the surface or deep in a sulcus of the brain. It is possible that this tumor represented a type of perivascular sarcoma of the brain (group A [?]), but the long postoperative period of survival would seem to operate against this conception. Without the benefit of postmortem examination, however, we did not think it possible to classify this neoplasm.

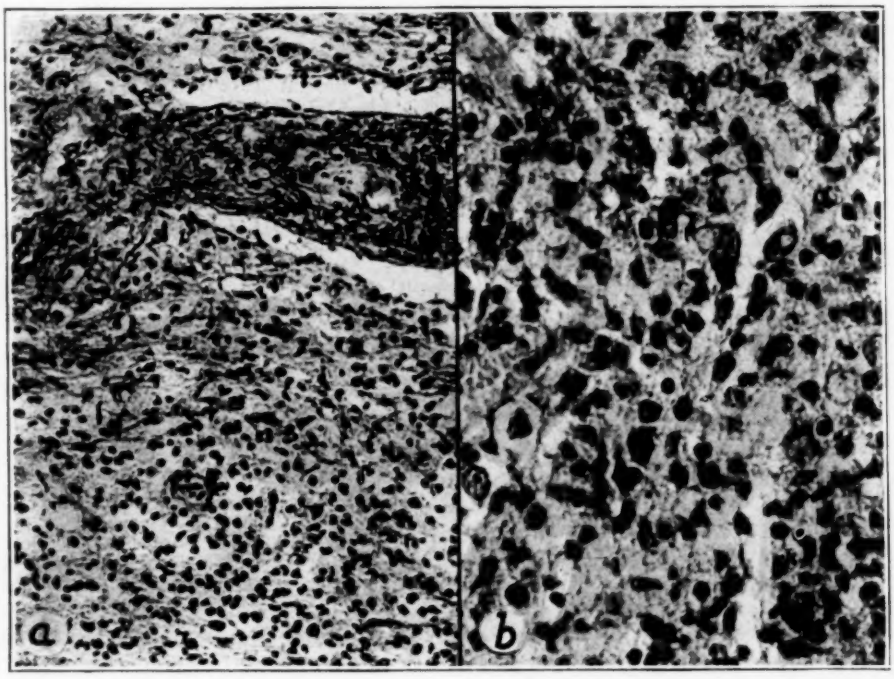


Fig. 9 (case 12).—Sarcoma of unknown type: (*a*) Fine reticulin fibers are here evident (Perdrau technic; × 200); (*b*) in this section are revealed cells of the type that were seen throughout the tumor (hematoxylin and eosin; × 435).

COMMENT

Much has been said about the variations and differences in the cells constituting primary sarcomas of the brain. Not a few authors have attempted by philosophic speculation to determine the embryonic origin of the meninges and the pial and cerebral vessels and their adventitia, and in this way to justify assignment of the development of all intracranial sarcomas to them. Such reasoning leads to the conclusion, on the one hand, that the tumors are of ectodermal origin (and therefore not sarcomas) and, on the other, that they are of mesodermal origin. Either conclusion is of little value in an embryologic sense, since no embryologist today clings to the outmoded theory of the specificity of the germ layers. A more logical approach is that of direct study of the histologic structure of these tumors,

and when this is carried out, these neoplasms all are found to be of connective tissue type; that is, they are sarcomas, regardless of the germ layer from which they supposedly arose. We have shown that primary sarcomas of the brain can be divided into fibrosarcoma, perivascular sarcoma and variations of these two types. However, when the two types just mentioned are closely studied, similarities between them appear, and the inclination is to group them together on the basis that they differ only in environment and rate of growth, although, to be sure, there are many differences which would prevent such a classification. Both types of tumors, as stated, arise from connective tissue. Whether the origin is from the connective tissue in the leptomeninges or from the connective tissue in or around the blood vessels (adventitia) has not as yet been proved, and the question does not appear to be of paramount histologic importance in the recognition and classification of these neoplasms.

The relation of fibrosarcoma to meningioma has been indicated in the fact that it is histologically identical with the malignant "stromal" (fibrous) type of meningioma, in the formation of which the arachnoid cap cell plays no part. This type of meningioma (malignant, fibrous or sarcomatous) differs from the primary fibrosarcoma in its gross appearance only in that it occurs on the surface of the brain and subsequently invades the underlying nerve tissue, whereas the primary fibrosarcoma occurs primarily within the brain.

The alveolar sarcoma described by Bailey[46] and by Hsü [7] was not encountered in our series, except in isolated small portions of 1 tumor, which was a fibrosarcoma. After a review of the published descriptions and photomicrographs in cases of the aforementioned neoplasm, we are impressed by its resemblance to the fibrosarcoma, to which the alveolar sarcoma may be closely related. This suggestion, however, does not alter the fact that it has a histologic resemblance to embryonic mesenchyma, from which it may arise.

Careful examination of the cases reported as instances of "perithelioma" has disclosed a heterogeneous group of neoplasms, among which were meningioma (angioblastic type), hemangioblastoma, ependymoma and even metastatic tumors, but there were no benign tumors or tumors of a low or a moderate degree of malignancy that apparently arose from the so-called perithelium. The only tumors of such a connective tissue origin (from the walls of intracerebral vessels) were true sarcomas of a high grade of malignancy, which we have chosen to call by the noncommittal term perivascular sarcoma, by which we have avoided argument in the matter of a special cellular covering of the intracranial vessels (perithelium).[47] Thus, the term "perithelioma" as used to designate a sarcomatous tumor of the intracranial structures seems to be relegated to disuse, just as has happened in modern pathologic investigations to the same term when used to denote neoplasms situated in other organs of the body.

SUMMARY

Twelve cases of tumors classified as "primary sarcoma of the brain" are reported. These lesions, arising in the brain as primary tumors, have been classified as fibrosarcoma (3 cases), perivascular sarcoma (7 cases) and sarcoma of unknown type (2 cases, in both of which the tumor probably belonged to one of the aforementioned two types).

---

46. Bailey.[4] Bailey and associates.[25]
47. Eberth.[11a] Scheinker.[34]

We have shown that fibrosarcoma may occur in any part of the cerebral hemispheres, but, strangely, we encountered none in the cerebellum. No valid explanation of our failure to find the lesion in the cerebellum can be made, except that the entire series may be too small for the point to be of significance. It is suggested that the lesions in the cases reported in the literature be divided into fibroma (benign), fibroblastoma (relatively benign) and fibrosarcoma (malignant); the histologic basis for this distinction is outlined. It is admitted that the fine point of distinction between the last two types in the case of a given tumor may at times be difficult. Variations in cellular patterns are discussed, and the occasional slight similarity between such tumors and the perivascular sarcoma is noted. The malignant fibroblastic, or "stromal," meningioma, which is lacking in the arachnoid cap cells, is considered histologically to be identical with the fibrosarcoma, but cases of this tumor were not included in our series. The fibroma and the relatively benign fibroblastoma, occurring within the brain, and the fibrous meningioma, occurring in the lateral, third and fourth ventricles, are also considered identical, since certain investigators have recently shown that they do not contain the arachnoid cap cell; hence, they differ from the fibrosarcoma herein discussed only in degree of differentiation.

Perivascular sarcoma is shown to vary in form from a local mass within the nerve tissue, surrounded by a pseudocapsule of degenerated and infiltrated brain tissue, to diffuse sarcomatosis involving large portions of the brain. We consider diffuse pial sarcomatosis to be another manifestation of this neoplasm, and an entity separate from the diffuse meningiomatosis, the latter being conceived to be of arachnoid (cap cell) origin, whereas the former is of connective tissue origin, probably arising from the pia mater.

Two unusual, unclassified tumors of proved sarcomatous nature are described. One was shown to resemble the malignant giant cell tumor (fibrosarcoma) of bone, and it is suggested that it be called a "malignant giant cell fibrosarcoma."

It is suggested that primary sarcomas of the brain probably originate from any connective tissue situated within the brain, that is, the blood vessels and their adventitia, or from the pia mater deep in the sulci, or from the lateral velum of the superior tela in the case of sarcoma of the lateral ventricle.

The Mayo Clinic.

DERMATOME HYPALGESIA ASSOCIATED WITH HERNIATION OF INTERVERTEBRAL DISK

J. JAY KEEGAN, M.D.
OMAHA

Herniated intervertebral disk has become recognized as the commonest cause of unilateral pain in the lower part of the back and of unilateral sciatic pain, on the basis of hundreds of cases in which the presence of the pathologic process has been verified at operation. Knowledge of the varying history and physical signs of this lesion has improved greatly as a result of the impetus given to the subject by Mixter and Barr [1] in 1934, through the application of intraspinal injection of iodized poppyseed oil and surgical methods to the demonstration of the lesion. In recent years there has developed increasing conservatism in the intraspinal use of iodized poppyseed oil, as a result of inflammatory and medicolegal complications, and greater emphasis has been placed on arrival at the diagnosis from the history and neurologic signs.[2] However, because of lack of anatomic definition, there is still considerable confusion and uncertainty in the interpretation of sensory and reflex loss from compression of a nerve root by a herniated intervertebral disk. It is the purpose of this paper to call attention to the definite and fairly constant dermatome loss of pain in the lower extremity from compression, stretching or section of a single nerve root, associated with herniation of a lower lumbar intervertebral disk, and to present a composite dermatome chart (fig. 10) for this hypalgesia, which follows a logical continuous serial distribution from the foot to the spine. The use of this chart in anatomic and neurologic textbooks should lead to better understanding of the subject than does the present confusion of illustrations.

The material on which this study is based was selected from 300 clinical cases of "low back and sciatic pain" which were carefully studied for neurologic signs during the past five years. A considerable number of these cases were atypical or did not present neurologic evidence which warranted the diagnosis of herniated intervertebral disk or operation. Many of the earlier cases with hypalgesia were not outlined carefully enough for one to recognize dermatome distribution. As neurosurgical experience in this field increased and the presence of single nerve root syndromes became recognized, skill in determining diagnostic dermatome areas of hypalgesia for localization of herniated intervertebral disks improved, and the use of myelograms taken with iodized poppyseed oil was discontinued, except in occasional atypical cases. A total of 146 instances of diagnostic dermatome hypalgesia have been found, in 78 of which the presence of the lesion was verified by operation, the diagnosis in the others remaining unverified because of early or mild symptoms not warranting surgical intervention. Of the 149 cases of dermatome hypalgesia, 90 were characteristic of involvement of the first sacral nerve root, 36 of a lesion of the fifth lumbar nerve root, 18 of a lesion of the fourth lumbar root, 1 of

From the Department of Surgery, Service of Neurological Surgery, University of Nebraska College of Medicine.

1. Mixter, W. J., and Barr, J. S.: Rupture of the Intervertebral Disc with Involvement of the Spinal Cord, New England J. Med. **211**:210 (Aug. 2) 1934.

2. Spurling, R. G., and Grantham, E. G.: Neurologic Picture of Herniations of the Nucleus Pulposus in the Lower Part of the Lumbar Region, Arch. Surg. **40**:375 (March) 1940. Dandy, W. E.: Concealed Ruptured Intervertebral Disks: A Plea for the Elimination of Contrast Mediums in Diagnosis, J. A. M. A. **117**:821 (Sept. 6) 1941; Improved Localization and Treatment of Ruptured Intervertebral Disks, ibid. **120**:605 (Oct. 24) 1942.

a lesion of the third lumbar root and 1 of a lesion of the second sacral root. Of the cases in which operation was performed, lesion of the first sacral nerve root was verified in 52, of the fifth lumbar nerve root in 16, of the fourth lumbar nerve root in 9 and of the second sacral nerve root in 1. Some cases of surgical stretching or section of single nerve roots, reported in the later part of the text as unquestionable lesions of a single nerve root producing typical dermatome hypalgesia, were included in this series and are shown in the table.

The most authoritative references for the dermatome, or segmental cutaneous distribution of nerve roots in man are the publications of Head [3] and of Foerster.[4] Head based his observations on a large number of cases of herpetic eruptions and the distribution of anesthesia with traumatic lesions of the spinal cord and the cauda equina. In Head's scheme (fig. 1) the cutaneous root areas overlap little and in this respect do not conform to the results of Sherrington's [5] physiologic experiments on the monkey, in which considerable overlap of root areas was found by a study of the sensibility in the area of the remaining single nerve root after section of the roots above and below it. Foerster applied Sherrington's method of root

Summary of Cases of Dermatome Hypalgesia

	Number of Cases
Total number of cases of "low back and sciatic" pain	300
Selected cases of dermatome hypalgesia	146
First sacral dermatome	90
Verified by operation	52
Verified by section of nerve root	4
Fifth lumbar dermatome	36
Verified by operation	16
Fourth lumbar dermatome	18
Verified by operation	9
Verified by section of nerve root	2
Third lumbar dermatome	1
Second sacral dermatome	1
Verified by root section	1
Total cases in which operation was done	78

isolation to man by section of the roots above and below a single root in enough cases to define most of the dermatomes in man. His results are illustrated in figure 2 and in Brock's [6] modification, shown in figure 3, for the lumbar and sacral dermatomes. Foerster found that the dermatomes in man overlap to the same degree as do those in the monkey and that, except for this greater overlap, their distribution in general corresponds with Head's.

The serial pattern of the dermatomes, as plotted by Head and by Foerster, is difficult to follow in the lower extremity because of the limitation of the fourth and fifth lumbar and the first sacral nerve root distribution to areas below the knee and the extensive overlap, shown by Foerster. However, it seems that a more evident serial pattern should be present, both in continuity from the foot to the spine and from the inner to the outer side of the foot, this assumption being based on the progressive extension of pain due to compression of a lumbar nerve root by a herniated intervertebral disk from the lower part of the back down the "sciatic" surface distribution on the buttock, thigh and leg to the foot and the known difference in nerve root distribution to muscles of the two sides of the foot. The chief

3. Head, H., and others: Studies in Neurology, London, Oxford University Press, 1920.

4. Foerster, O.: The Dermatomes in Man, Brain **56**:1 (March) 1933.

5. Sherrington, C. S.: Experiments in the Peripheral Distribution of the Fibers of the Posterior Roots of Some Spinal Nerves, Phil. Tr. Roy. Soc., London **184**:641-763, 1893.

6. Brock, S.: The Basis of Clinical Neurology, Baltimore, William Wood & Company, 1938.

deterrent to recognition of sensory loss in a single nerve root dermatome has been the statement by Foerster [4] that "the amount of overlap of the different dermatomes is so considerable that division of a single nerve produces no loss of sensibility." Consequently, when sensory loss has been observed in cases of lesions of nerve roots supplying the lower extremity, it has been assumed that more than one nerve root must be involved, and careful plotting of the hypalgesic area has not been done. It should be called to attention that Foerster qualified the foregoing statement by adding, "at least [as detected] by the usual clinical methods of examining sensibility." He did recognize that sensory diminution can be demonstrated "by counting carefully the number of pressure and pain spots in the area innervated

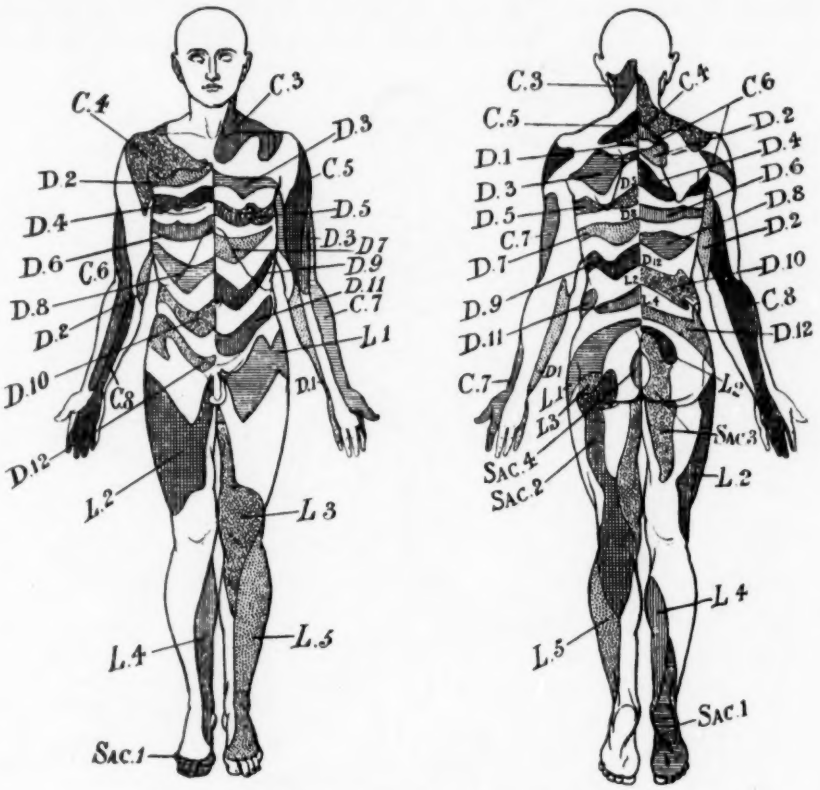


Fig. 1.—Head's chart of the dermatomes in man.

by that root, and by measuring exactly the threshold of the spots for mechanical and electrical stimuli." Thus it was recognized by Foerster that there are possibilities of detecting sensory loss from a lesion of a single nerve root.

The method of testing for this sensory loss used in the study present is not difficult, and it is hard to understand why others have not made these observations before. In the beginning tests were made for pain, touch and temperature sense in the usual manner, but it soon was learned that the positive response to pain stimuli could be differentiated in degree more accurately than the response to touch or temperature stimuli by the average patient. Also, there is less confusion in pain sense due to varying reactions referable to hair follicles and thickened skin and less overlap of pain dermatomes than of touch dermatomes. The simplest and most convenient test for pain sense, that of light pinprick, was found satisfactory. This test undoubtedly could be refined in accordance with quantitative electrical methods,

but for clinical and practical purposes this is not necessary. The patient must be cooperative and clearly understand that the test is for slight reduction, and not for complete loss, of pain sense. This is established by the examiner's comparing similar areas on the two feet until an area is found where pain is "not quite so sharp." Then the two sides of that foot are tested, a similar response of difference being obtained, and the pinprick is passed rapidly from the slightly hypalgesic zone across the foot until a "natural" or a sharper zone is entered. The region of most definite loss usually is in the distal part of the foot or leg, in agreement with Head's and Foerster's demonstration of root areas in these regions alone. If one keeps in mind the expected dermatome distribution and always starts in the hypalgesic zone, it is surprising how constantly the patient will record the transition line and how accurately the dermatome loss will correspond in different patients with proved lesions of single nerve roots. Not all single nerve root syndromes due to herniated

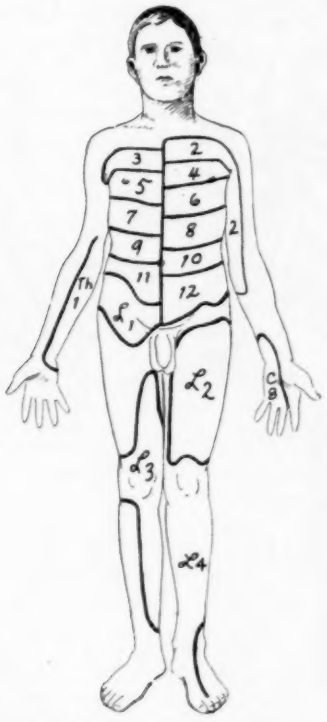


Fig. 2.—Foerster's chart of the dermatomes in man.

disk can be traced the full length of the dermatome, for in many cases there is only intermittent pressure on the root, without complete loss of nerve function. When there is complete loss of the ankle or the knee jerk from a lesion of a disk, a complete dermatome area of hypalgesia from the foot to the spine usually can be demonstrated for either the first sacral or the fourth lumbar nerve root. Other indicative signs are the presence of subjective numbness and tingling sensations, usually located accurately within the dermatome involved. Subjective pain is not so reliable a guide to dermatome hypalgesia and may be represented only by hyperalgesia, although the distribution of the pain often is suggestive of the root involved.

LESIONS OF THE FIRST SACRAL NERVE ROOT

The first sacral nerve root furnishes the best example of isolated compression of a nerve root by a small discrete herniation of the nucleus pulposus, in this case

that of the fifth lumbar intervertebral disk. Such a herniation occurs in over 50 per cent of all cases of herniated disk,[7] and hence the syndrome is encountered frequently. The anatomic relations of this nerve root should be understood before one proceeds to interpretation of the pathologic changes, as they differ somewhat from those of other nerve roots of this region (fig. 4). The first sacral nerve root is rather long in its extradural course within the spinal canal. It leaves the main

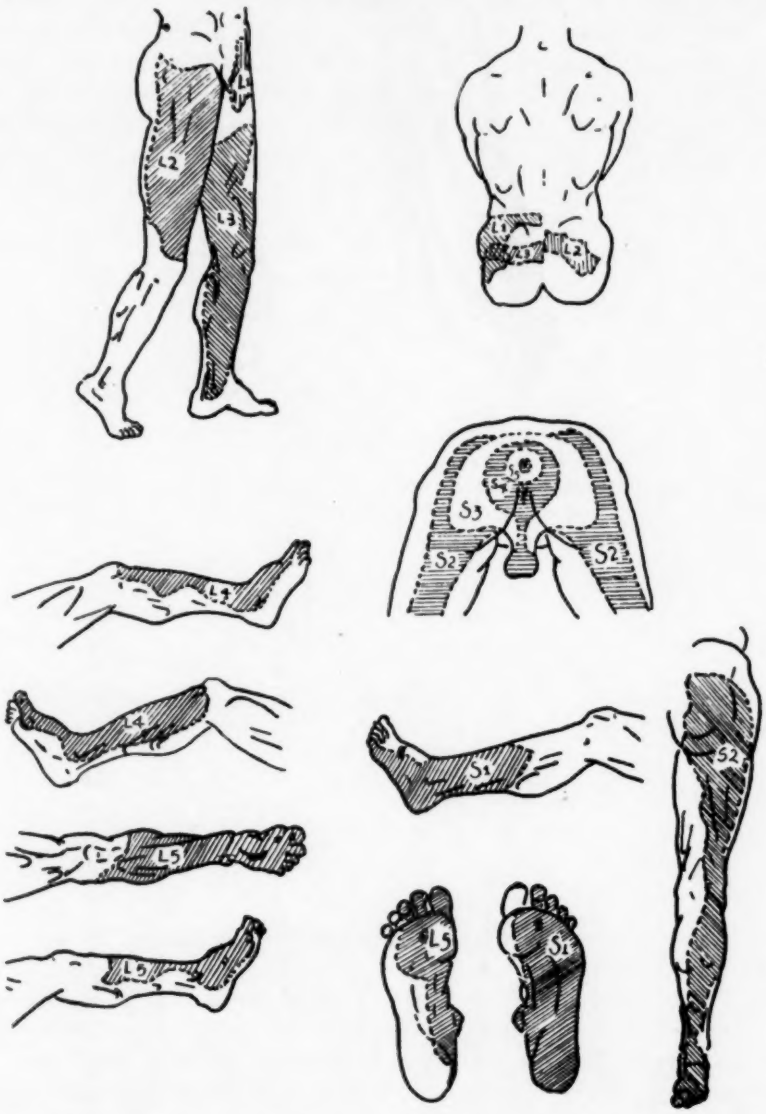


Fig. 3.—Brock's modification of Foerster's charts of the lumbar and sacral dermatomes.

dural canal well above the fifth lumbar intervertebral disk, crosses this disk in the lateral angle of the narrowed intraspinal space of this region, has its sensory ganglion located below the disk, beneath the first sacral lamina, and leaves the spinal canal through the first sacral foramen. The length of this portion of the first sacral nerve root is 3 to 4 cm. Herniation of the nucleus pulposus of the fifth

7. Love, J. G., and Walsh, M. N.: Intraspinal Protrusion of Intervertebral Disks, Arch. Surg. **40**:454 (March) 1940.

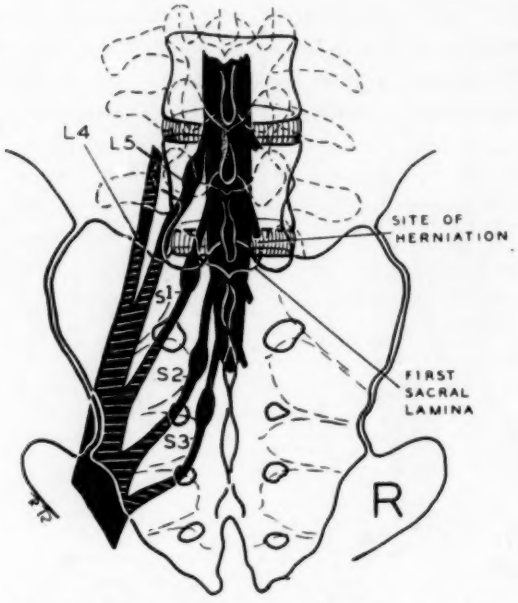


Fig. 4.—Drawing made over a roentgenogram, showing the relation of the first sacral and fifth lumbar nerve roots to the intervertebral disks.

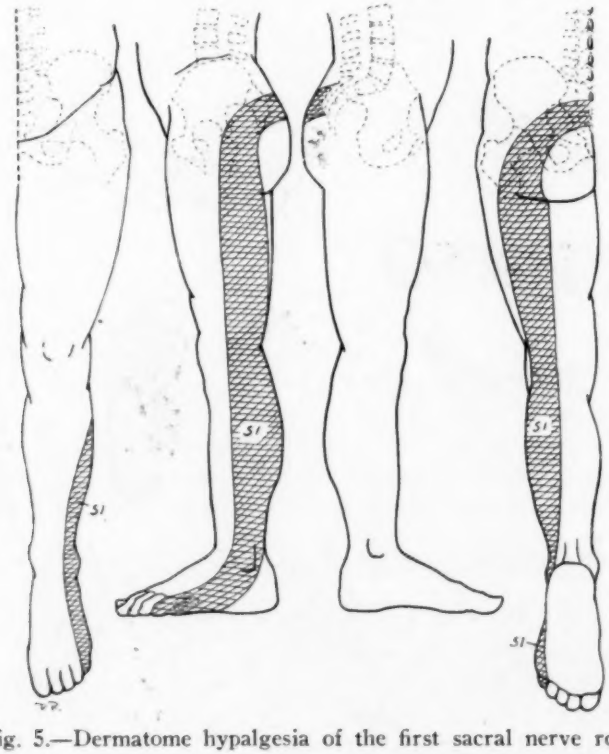


Fig. 5.—Dermatome hypalgesia of the first sacral nerve root.

lumbar intervertebral disk usually occurs intraspinally directly beneath or slightly medial to this nerve root. Such a herniation rarely is over 1 cm. in diameter and compresses the nerve in the narrow posterolateral angle of the spinal canal against the overlying ligamentum flavum and the first sacral lamina. On surgical exploration the flattened and adherent first sacral nerve root is separated with some difficulty and retracted medially to expose the easily palpable discrete herniation of the disk. No other nerve root is seen in the dissection, and it seems necessary to interpret the symptoms and hypalgesia in such a case as due entirely to involvement of this root. Further evidence of this fact has been obtained several times by rechecking the area of hypalgesia after the nerve root has been stretched to considerable degree at operation, when a more definite complete dermatome distribution may be found, whereas before operation only the distal portion of the dermatome

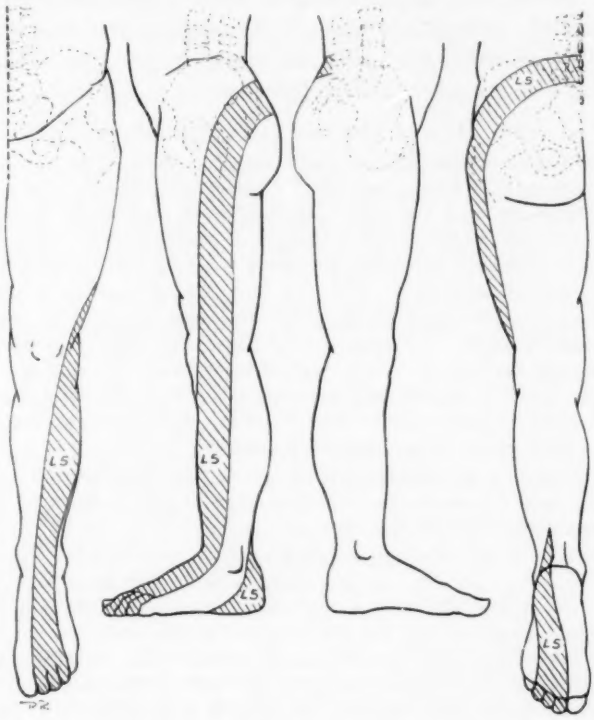


Fig. 6.—Dermatome hypalgesia of the fifth lumbar nerve root.

could be demonstrated. It is appreciated that some herniations include the annulus and spread more widely than the common early herniation of the nucleus pulposus and that more than one nerve root may be involved. However, this is not the rule, and even in cases of such a lesion careful study of the differing degrees of dermatome hypalgesia will identify the nerve root chiefly and primarily involved. Case 5 is such an instance and is presented as an example of a lesion of the second sacral nerve root.

The dermatome area of hypalgesia thus outlined for the first sacral nerve root (fig. 5) is remarkably constant in its outer limits and varies chiefly in its degree of extension upward to the sacral portion of the spine, the latter being dependent on the severity of compression of the root. The most constant and definite area of hypalgesia is found on the outer surface of the foot and calf. Its medial boundary ends sharply between the fourth and the fifth toe on both the dorsum and the sole of the foot. It turns across the lateral border of the foot in front of the heel and

then passes over the external malleolus. In the leg and thigh it occupies a posterolateral position, with the medial border about at the midline. It curves around the midgluteal region to the upper sacral region of the spine. The width averages about 8 cm. but is somewhat greater on the calf and is narrowed, or less distinct, back of the knee. In cases of the complete first sacral dermatome syndrome, the ankle jerk is absent, the knee jerk is normal and a small area of analgesia may be found on the lateral border of the foot and calf (fig. 9). A typical clinical example of the first sacral nerve root syndrome is presented (case 1). This is one of 90 cases in which herniation of the fifth lumbar intervertebral disk was diagnosed on the finding of an area of hypalgesia on the foot and ankle corresponding to the first sacral dermatome; in 52 of these cases the diagnosis was confirmed at operation. In case 1 confirmation was obtained at operation by observation of a discrete herniation compressing only the first sacral nerve root, and after operation by the finding of a complete dermatome area of hypalgesia extending from the foot to the spine, caused by stretching of the first sacral nerve root at operation, the area of hypalgesia disappearing within three weeks.

CASE 1.—Mr. J. J., aged 31, was admitted to St. Joseph's Hospital.

History.—"Sciatic" pain developed on the left side without recognized trouble with the back. The pain extended from the left "hip" (superior gluteal) region posterolaterally to the calf and the lateral part of the ankle, with a sensation of numbness of similar distribution. The patient continued to do light work for about four months but at times had to lie down to get relief. Finally, after he had tried various treatments, he became completely disabled.

Examination.—Examination on June 8, 1942 showed reduction of the ankle jerk on the left side, greatly limited ability to raise the straight left leg and pain on pressure over the left lumbosacral junction and on jugular compression. A definite area of hypalgesia corresponding to the first sacral dermatome was found on the lateral surface of the foot, ankle and calf and a less distinct area over the thigh and buttock (fig. 5). On the lateral border of the foot there was a small area of analgesia.

Diagnosis.—The diagnosis was herniation of the nucleus pulposus of the fifth lumbar intervertebral disk, with compression of the left first sacral nerve root. Surgical treatment was recommended, without myelograms being taken.

Operation (July 28).—The left ligamentum flavum of the fifth lumbar intervertebral space was removed, and a small discrete cartilaginous tumor was observed on the fifth lumbar intervertebral disk, over which the first sacral nerve root was stretched tightly and flattened and to which it was adherent. Sharp dissection was necessary to free the nerve and to retract it medially to expose the tumor of the disk. An incision into the tumor released the fibrocartilage of the nucleus pulposus, which was grasped with a clamp and removed in one piece, part of which came from the disk proper, and which was thought to be the entire nucleus pulposus. This completely reduced the tumor and permitted the rather long first sacral nerve root to return to a normal position. There was no evidence of contact of any other nerve root with the tumor.

Progress.—There was immediate relief of all the old pain in the leg, this pain being accurately differentiated from the soreness of the wound, which lasted about five days. The patient was able to be up without support in two weeks and returned to light work in one month. After operation the dermatome hypalgesia in the first sacral dermatome was more definite than before operation, owing to stretching of the nerve, and was outlined accurately with light pinprick, as shown in figure 5.

LESION OF THE FIFTH LUMBAR NERVE ROOT

The next commonest herniation of an intervertebral disk is that of the fourth lumbar, the lesion compressing the fifth lumbar nerve root. The syndrome of this root is characterized by pain which radiates slightly more to the front of the leg, and there may be a subjective sensation of numbness which the patient locates on the top of his foot or middle toes. The ankle or the knee jerk may be reduced but is never completely absent. The dermatome area of hypalgesia for this nerve root is more difficult to outline than that for the first sacral nerve root. However, at

times a definite area of hypalgesia can be found (fig. 6) extending from the dorsum of the foot, including the three middle toes, obliquely up over the anterolateral aspect of the leg and the lateral part of the knee and thigh, to curve around the superior gluteal region to the lumbosacral junction. The best example in this study of the syndrome referable to this nerve root is a clinical case in which no diagnostic dermatome area of hypalgesia could be found before operation but in which surgical exploration disclosed a typical herniated nucleus pulposus of the fourth lumbar intervertebral disk beneath the left fifth lumbar nerve root. Owing to the rather wide dural canal and the short fifth lumbar nerve root at this level, considerable traction on the nerve was necessary to expose and remove the herniation. After operation the patient complained of numbness on the top of her foot and middle toes, and careful plotting of the area of hypalgesia showed the full dermatome (fig. 6). Of special interest was an area of hypalgesia on the sole of the foot, which extended posterolaterally to include the lateral surface of the heel and to end in a point on the lateral side of the tendo Achillis. Of the 36 cases of the syndrome referable to the fourth lumbar intervertebral disk, the presence of the lesion was proved by operation in 16.

CASE 2.—Miss J. S., aged 17, was admitted to the Nebraska Methodist Hospital.

History.—The patient presented a rather atypical history of pain in the lower part of the back and radiating down the left leg, which developed gradually, without any definite neurologic signs to locate the lesion of the nerve root except slight reduction of the ankle jerk on the left side and a questionable area of slight hypalgesia on the middorsum of the foot and toes, suggesting involvement of the fifth lumbar nerve root. In addition, she had considerable congenital abnormality of the lumbosacral portion of the spine. She did not improve satisfactorily after eleven months of conservative orthopedic treatment, and an air myelogram was recommended for better diagnosis, but permission was refused. Because of the suspected herniation of the fourth lumbar disk, it seemed a better policy to explore this space before a contemplated operation for spinal fusion was undertaken.

Operation.—On Dec. 4, 1942, with the patient under general anesthesia, a herniated nucleus pulposus was found on the left side of the fourth lumbar disk, beneath a very short fifth lumbar nerve root. Considerable traction on this root was necessary to expose and remove the herniation, and the comment was made that this stretching was likely to cause temporary loss of function of the nerve root. After removal of the disk spinal fusion with use of a tibial graft was done by Dr. Robert Schrock.

Progress was satisfactory, with the patient in a bivalved body cast. She volunteered the information after operation that there was numbness on the top of her left foot and middle toes and expressed concern that this might persist. Careful tests with light pinprick outlined a definite and complete dermatome strip of hypalgesia for the fifth lumbar nerve root, extending from the middle three toes to the lower lumbar portion of the spine, with a corresponding wedge-shaped area on the sole of the foot (fig. 6). Within two weeks this sensation of numbness and dermatome area of hypalgesia had disappeared, an indication that it was due to traction on the one nerve root.

Progress.—The patient has been relieved of the pain in her leg and the lower part of her back.

LESION OF THE FOURTH LUMBAR NERVE ROOT

The syndrome of compression of the fourth lumbar nerve root is fairly characteristic and is easily differentiated from the much more common syndrome of the first sacral nerve root. The accompanying pain typically radiates in spiral manner over the knee cap and the medial part of the tibia to the great toe, with paresthesia or a sensation of numbness in this distribution over the anteromedial aspect of the leg and the great toe. In the complete syndrome the knee jerk is absent and the ankle jerk is normal. Several cases of the fourth lumbar dermatome type of hypalgesia due to herniation of the third lumbar intervertebral disk were found, but the best example was that of an elderly man (case 3) in which surgical exploration disclosed a very small neurofibroma of the fourth lumbar nerve root and the nerve was cut for removal of the tumor. Thus, unquestionable dermatome hypalgesia due

to involvement of a single nerve root was demonstrated, in agreement with the cases of herniated disk (fig. 7). The area of hypalgesia for this nerve root constantly includes the great toe and the medial border of the foot in front of the heel, then curves over the internal malleolus and extends up the inner surface of the tibia, over the patella and the anterolateral aspect of the thigh and around the ileum to the lower lumbar portion of the spine. Of the 18 cases in which this dermatome area of hypalgesia was presented, the lesion was verified at operation in 9.

CASE 3.—Mr. R. B. O., aged 68, was admitted to the Methodist Hospital.

History.—The patient complained of a steady aching pain in his left leg of two years' duration, with no history of trouble in the lower part of the back. The pain extended from the left "hip" region and anterolateral portion of the thigh over the knee and the inner aspect of the leg and ankle, and at times was associated with a sensation of numbness or

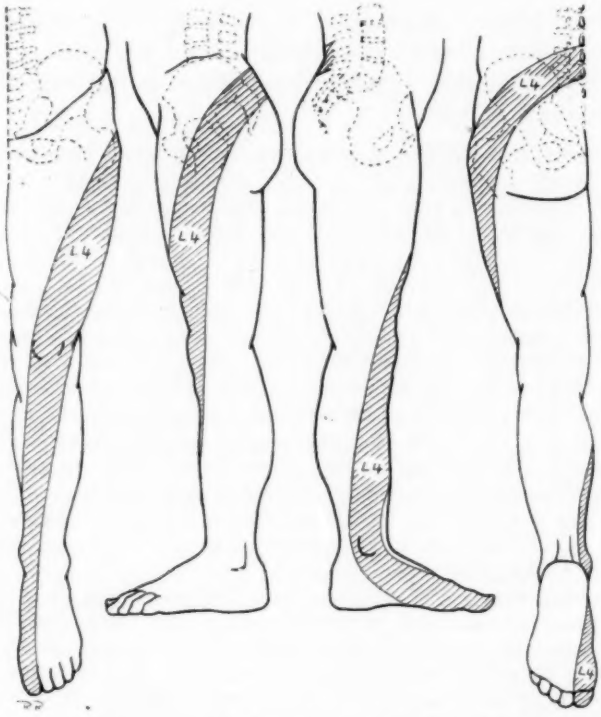


Fig. 7.—Dermatome hypalgesia of the fourth lumbar nerve root.

of being "asleep," which he located by passing his hand in spiral manner along the course of the pain.

Examination.—Straight leg raising was not limited, but the knee jerk was reduced on the left side, and a spiral dermatome strip of hypalgesia was outlined, as shown in figure 7, which was identified as characteristic of the fourth lumbar nerve root. Roentgenograms of the lumbar portion of the spine showed nothing diagnostic.

Diagnosis.—The diagnosis was a discrete lesion of the left fourth lumbar nerve root, probably not herniated disk, but a condition which warranted exploration.

Operation.—On Dec. 7, 1942, with the patient under anesthesia induced with avertin supplemented by local anesthesia, the lamina of the left fourth lumbar vertebra was removed. No herniation was seen at either the third or the fourth intervertebral disk. However, a small, firm nodule was felt within the dural sheath of the fourth lumbar nerve root at its junction with the main dural canal. The dura was opened over this nodule, and a small, firm tumor, about 5 mm. in diameter, was observed on the nerve just before it entered the nerve sheath proper. At first this was thought to be an abnormally placed dorsal root ganglion, but it seemed too firm and was removed by cutting the attached nerve root on each side, an underlying

uninvolved portion, judged to be the motor root, being left. Microscopic diagnosis of this tumor was neurofibroma, no ganglion cells being present.

Progress.—The patient was completely relieved of his long-standing pain in the leg and at first observed that the old numbness, or sensation of being "asleep," was gone; however, when he passed his hand over his leg, he commented that the inner area of the leg seemed more "dead" than before. In a recheck on the area of hypalgesia there was outlined more definitely than before a typical spiral dermatome strip for the fourth lumbar root, extending from the great toe to the lumbar portion of the spine, with an oval area of analgesia on the medial surface ot the tibia (fig. 7). The knee jerk was absent; the ankle jerk was normal.

LESION OF THE THIRD LUMBAR NERVE ROOT

Herniation of the second lumbar intervertebral disk, which would compress the third lumbar nerve root, is rather uncommon, and the only instance of this type of dermatome hypalgesia is a clinical case in which the causative lesion was verified

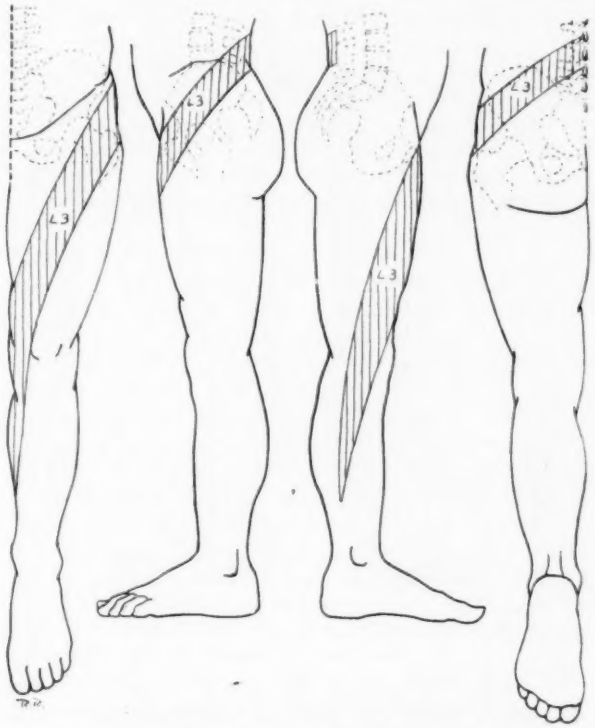


Fig. 8.—Dermatome hypalgesia of the third lumbar nerve root.

only by roentgenogram. The pain began in the left upper lumbar region and radiated around the crest of the ileum to the anteromedial aspect of the thigh and the inner surface of the knee. The knee jerk was normal. The area of hypalgesia was a typical spiral dermatome strip, which extended from the inner aspect of the mid-leg over the inner side of the knee, obliquely across the anterior surface of the thigh and around the crest of the ileum to the midlumbar portion of the spine (fig. 8).

CASE 4.—Mr. J. L., aged 63, was admitted to the Lutheran Hospital.

History.—On Jan. 30, 1943, pain first developed in the left upper lumbar region. He associated the onset with his having chipped ice off the sidewalk about a week before, although no special pain developed then. The pain in the back was gradual in onset and tended to extend across his back and to affect his right hip (crest of the ileum). Radiating pain in the left leg developed rather suddenly a few days later; it was somewhat like a toothache, with jabs in the anterior surface of the thigh and the inner surface of the knee cap and leg down

to the midregion. Numbness developed in the iliac region when he lay on his left side, and some aching pain was located in the left groin, which seemed to extend through medially to the buttock, over the tuberosity of the ischium. He obtained most relief by lying on his left side, with the left leg drawn up and the right leg extended out straight and backward. In walking he could not bring the left thigh forward well because of pain and loss of power.

Examination.—He was a rather small man, weighing 125 pounds (56.7 Kg.), who used crutches to walk. The knee and ankle jerks were active and equal on the two sides, and straight leg raising was moderately limited on the left. Sensory tests with light pinprick outlined a definite spiral strip of slight hypalgesia (fig. 8) extending from the medial side of the left knee obliquely across the anterior portion of the thigh, over the anterior crest of the ileum to the midlumbar portion of the spine. Roentgenograms of the spine at this time showed no tumor, but later roentgenograms showed a discrete metastatic tumor involving the lower portion of the second lumbar vertebra on the left side.

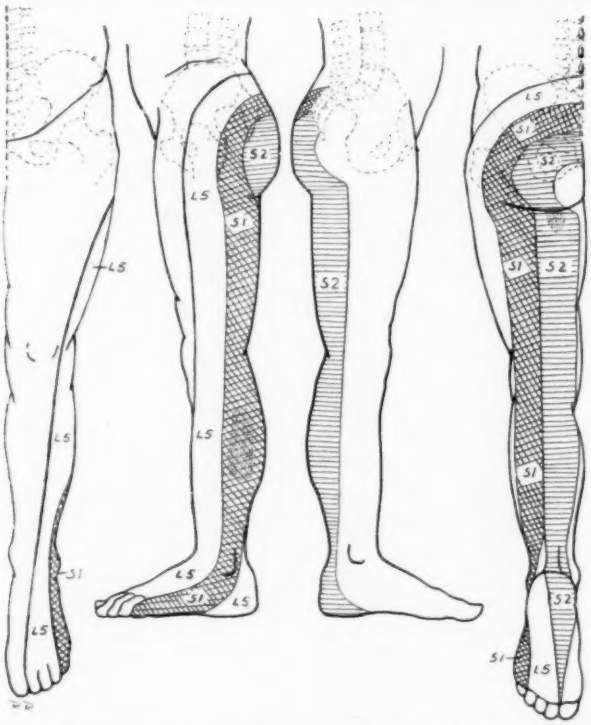


Fig. 9.—Dermatome hypalgesia of the first sacral, second sacral and fifth lumbar nerve roots in a case.

Diagnosis.—The diagnosis was compression of the left third lumbar nerve root by metastatic tumor. Operation was not recommended because of the character of the lesion.

LESION OF THE SECOND SACRAL NERVE ROOT

The second sacral nerve root is not involved by herniated intervertebral disk unless the herniation is so great that several sacral nerves are compressed, and then the lesion is difficult to differentiate. The opportunity to outline the dermatome hypalgesia of the second sacral nerve root was obtained in an unusual case in which there were six lumbar vertebrae and the nerve root overlying the last, or sixth, lumbar disk was cut for relief of pain, on the assumption that it was the first sacral nerve root. The dermatome of hypalgesia thus outlined (fig. 9) had its greatest loss of pain sense in the lower part of the buttocks and the upper posterior region of the thigh, noted by the patient as subjective numbness, with a small area of analgesia at the gluteal fold. Distally, the area of hypalgesia extended along the

posteromedial surface of the thigh and leg and over the inner surface of the heel, so that the space left between the fourth and the fifth lumbar dermatome on the sole of the foot was filled in. This case is presented in some detail because its course was unusually complicated and it illustrates the accuracy of interpretation of involvement of multiple nerve roots from dermatome hypalgesia, with verification by nerve root section.

CASE 5.—Mr. J. L. T., aged 38, was admitted to the Nebraska Methodist Hospital.

History.—Sudden pain developed in the lower part of the back on May 26, 1942, when the patient slipped while lifting a 100 pound (45.4 Kg.) sack. He felt a snap in the lower part of his back, and both legs went out from under him; he was unable to stand or straighten his back because of pain, which radiated into the "hip," the posterior portion of the thigh and the lateral aspect of the calf on the left. He was treated with rest in bed and traction, which seemed to make the pain worse. The lateral surface of his left ankle and foot seemed cold to him, and a burning sensation developed. Coughing and sneezing increased the pain, and he had to be careful in turning in bed or in sitting up in order to protect the lower part of his back.

Examination (June 27, 1942).—He was a tall, slender man, of reddish complexion. Straight leg raising was greatly restricted on the left side and caused pain in the left lower portion of his back and down his left leg to the lateral part of the calf and ankle. Tenderness to pressure was located over the left side of the lumbosacral junction, and the ankle jerk was reduced on the left. Tests for pain sensation in the left leg outlined a fairly definite first sacral dermatome strip of hypalgesia, extending from the lateral border of the foot over the external malleolus, the lateral portion of the calf, posterior part of the thigh and the superior gluteal region to the lower lumbar portion of the spine (fig. 9).

Diagnosis.—The diagnosis was intraspinal herniation of the nucleus pulposus of the fifth lumbar intervertebral disk on the left side, compressing intermittently the first sacral nerve root.

Recommendation.—Continuation of conservative rest and immobilization was advised.

Progress.—Traction of the left leg and a brace for the lower part of the back were tried, without relief except when the patient was in bed. Pain in the lower part of the back continued when he was up, with subjective numbness and objective hypalgesia on the lateral border of the foot. On July 15 neurosurgical treatment was recommended.

First Operation (July 20, 1942).—With the use of local anesthesia, the left ligamentum flavum between the last lumbar and the first sacral vertebra was removed; no involvement of the nerve root by a herniated disk was seen in this interspace. The ligamentum flavum next above, thought to be in the fourth lumbar interspace, but in reality in the fifth, was removed, and a typical discrete herniation of the nucleus pulposus, compressing the nerve root below, was observed. This was satisfactorily removed in one large piece, which was judged to be the entire nucleus pulposus. No bone was removed.

Progress.—The expected relief from the pain in his left leg did not occur, although the pain in his back was better. A recheck on August 17 showed a definite dermatome strip of hypalgesia corresponding to the area previously defined for the left first sacral nerve root (fig. 9); this finding suggested that something had been missed on the last, presumably the fifth, lumbar intervertebral disk. There were a subjective sensation of numbness over the posterolateral surface of the thigh and radiating pain into the lateral portion of the calf, ankle and toes, with a burning sensation in the lateral border of the toes. The ankle jerk was reduced on the left side. On August 31 a fairly definite strip of hypalgesia was found medial to the area of hypalgesia previously defined for the first sacral dermatome, indicating added involvement of the fifth lumbar nerve root above (fig. 9). Because of the unsatisfactory progress and the definite neurologic signs, a second operation was recommended, with the idea of sectioning the sensory portion of the first sacral nerve root to relieve the pain so definitely located in this dermatome.

Second Operation (October 8).—Local anesthesia was induced with procaine. Nothing was seen on the nerve root crossing the last lumbar intervertebral disk, but the dorsal two thirds of this root was cut. The next disk above was rough and presented some wide protrusion where the herniated nucleus pulposus had been removed previously, with the overlying nerve somewhat adherent and flattened. Protruding tissue was removed from this disk.

Progress.—The patient continued to have pain radiating into the left hip, thigh, leg and foot, as before operation, but he now had an area of subjective numbness located over the medial part of the left gluteal fold, identified by tests for hypalgesia as in the distribution

of the second sacral nerve, not the first sacral nerve, which presumably was cut at operation. Roentgen examination of the lumbar portion of his spine at a higher level then disclosed that he had six well formed lumbar vertebrae; thus, the nerve cut was identified as the second sacral nerve root and the original herniated nucleus pulposus as the fifth lumbar, with the first sacral nerve involved over it. It now seemed possible that there was another herniation at the true fourth lumbar disk which had not been explored, although the main distribution of pain, subjective numbness and objective hypalgesia still was in the first sacral dermatome. There was a considerable area of analgesia in the lateral portion of the calf (fig. 9). Recognition of the misinterpretation of the nerve roots at previous operations and the patient's continuing complete disability led to recommendation of a third operation, this time to explore the true fourth lumbar disk, to cut the true first sacral nerve root, if still involved by a protruding fifth lumbar disk, and to do a spinal fusion operation for added stabilization of the back. The fusion operation was postponed because of chronic infection of the respiratory tract.

Third Operation (Jan. 22, 1943).—With the use of local anesthesia induced with procaine, the lamina of the left fifth lumbar vertebra was removed; no herniation was observed at the fourth intervertebral disk, but the fifth lumbar nerve appeared to be involved in the intervertebral canal by a wide protrusion of the spongy and unstable annulus of the fifth lumbar intervertebral disk. The first sacral nerve root was densely adherent to this protrusion; it was dissected free with difficulty and was cut well above the disk. The sensory and motor portions could not be separated.

Progress.—The patient has been free of his old pain in the left leg since the last operation and was able to be up with comfort two weeks after the operation.

COMMENT

The preceding observations on definite dermatome loss of pain associated with isolated lesions of each of the third, fourth and fifth lumbar and first and second sacral nerve roots permits the drawing of a fairly complete composite dermatome chart for the lower extremity and accurate identification of the syndrome of each nerve root.

This dermatome chart (fig. 10) represents the areas of slight, but definite, reduction of pain sensation following compression, stretching or section of single nerve roots, but does not indicate the full dermatome distribution. Also, the chart does not show some apparent overlap of the dermatomes above the knee, as plotted by my method. Below the knee, and particularly in the foot, there is little or no overlap of areas, in accordance with the observations in many clinical cases of herniated intervertebral disk. This makes their occurrence diagnostic of the nerve root involved and thus localizes the herniation in the disk next above. The absence of significant overlap of these dermatomes of hypalgesia suggests that they represent rather accurately the major, or primary, innervation of each nerve root and that there is an additional lesser, or secondary, innervation which overlaps adjoining nerve areas but is not detectable with an intact adjoining primary innervation.

Extension of these dermatomes of hypalgesia the full length, from the foot to the spine, may be questioned, as this presents the most radical disagreement with the observations of Head and Foerster. However, such extensions certainly can be found in cases of complete loss for a single nerve root by any one who tests for them carefully with the patient's understanding and cooperation. The dermatomes are more difficult to outline above the calf, particularly back of the knee and in the gluteal region, both because of lesser representation there and because of the incomplete compression of the nerve root often presented by herniated intervertebral disk. Yet this continuity is in accordance with the common progressive downward sequence in development of pain from the superior gluteal region to the foot with irritation of nerve roots, which indicates an anatomic representation of pain sense the full length of the dermatome.

Another point of disagreement with Head's and Foerster's charts is the definite serial distribution of the fourth and fifth lumbar and first sacral dermatomes in the foot from the inner to the outer border, including the plantar surface. Head's inclusion of all the toes in the first sacral dermatome seems contrary to the anatomic distribution and was not confirmed by Foerster's observations. In fact, Foerster did show definite division lines between three dermatomes on the dorsum of the foot, the fourth lumbar dermatome including only the great toe and the first sacral

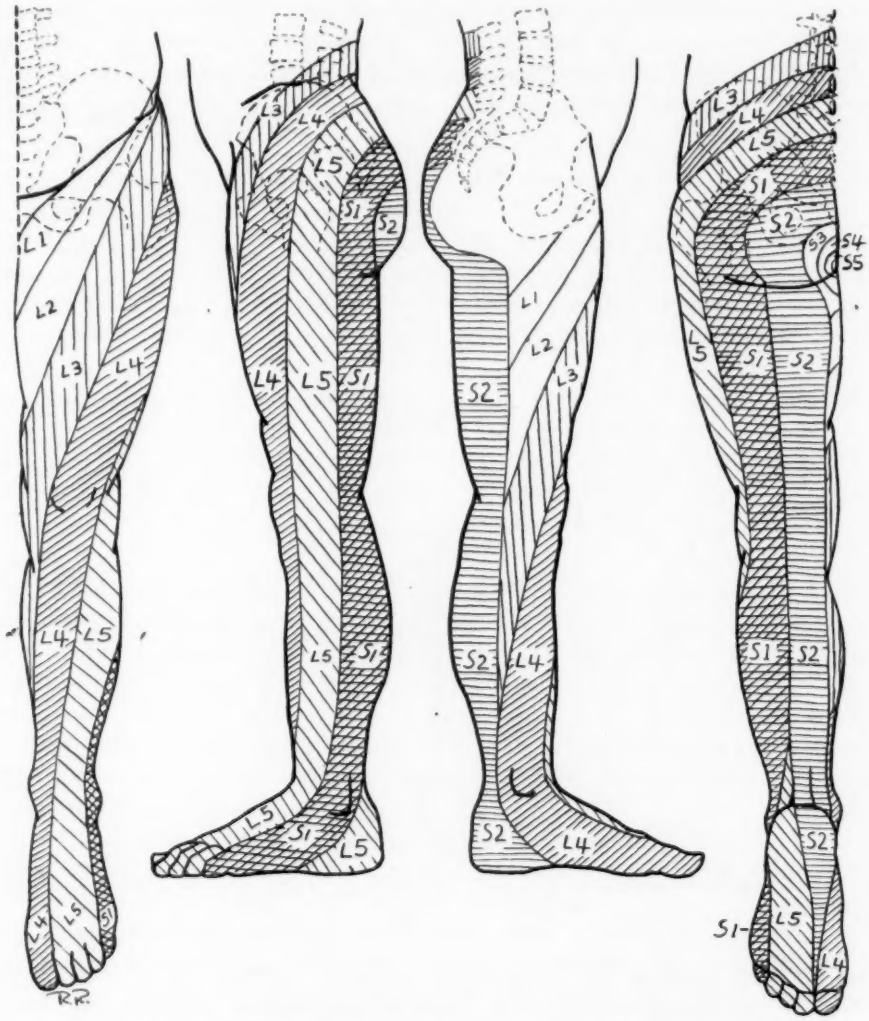


Fig. 10.—Composite dermatome chart of the lower extremity, determined by outlining the area of hypalgesia due to lesion of a single nerve root.

dermatome including only the little toe, with the intervening space supplied only by the fifth lumbar nerve root. This distribution is illustrated by a composite drawing (fig. 11) of these three dermatomes taken from Foerster's charts, which shows the same pattern in the foot as that observed in this study, except that a much greater overlap of the fifth lumbar dermatome was found by his method. The somewhat bizarre, but definite, pattern of the dermatomes of hypalgesia on the sole of the foot and heel is interesting, with four nerve roots represented from the medial to the lateral side, in the order of the fourth lumbar, the fifth lumbar, the second

sacral and the first sacral, with the heel covered by the fifth lumbar and the second sacral dermatome. The opportunity to plot these two central areas was unusual, and the recordings were satisfactory, owing to absence of callus from prolonged disuse; the pattern, moreover, is reasonable in consideration of the expected extension of the fifth lumbar nerve supply through the midfoot.

An important value of this composite dermatome chart for the lower extremity is the furnishing of a more easily understood anatomic chart of the dermatomes. Reference to anatomic and neurologic textbooks and monographs shows great variation in the dermatome charts used in illustration, most being frankly diagrammatic and omitting the lines in the foot and toes. Reproduction of Head's and Foerster's charts has not helped much in portrayal of the serial pattern of the dermatomes in the lower extremity because of their interrupted and overlapping distribution.

Fig. 11.—Composite dermatome chart of the dorsum of the foot, modified from Foerster's dermatome areas.

Although it is recognized that the composite chart presented by this study may be inaccurate in that it represents not the complete dermatome of remaining sensation, as studied by Foerster, but rather the dermatome of reduction of pain sense from loss of a single nerve root, and does not show some evident overlap in the proximal portion, yet it is reasonably accurate and in accordance with neurologic observations. It seems better for use in anatomic and neurologic textbooks than the illustrations in present use.

A final observation pertains to isolated compression of the first sacral nerve root by a small, discrete herniation of the nucleus pulposus of the fifth lumbar intervertebral disk. This is by far the commonest lesion causing unilateral "low back and sciatic pain." The common, rather vague, clinical interpretation of this "sciatic" pain without careful plotting of the associated sensory loss and exact knowledge of the anatomic course and dermatome distribution of the lower lumbar and first

sacral nerve roots can no longer be justified. This applies particularly to the first sacral nerve root, which has an entirely intraspinal course until its emergence through the first sacral foramen, well below the lumbar portion of the spine (fig. 4). When the syndrome of involvement of the first sacral nerve root is found, it is not reasonable to try to explain it on any basis other than that of an intraspinal lesion which interferes with this nerve root alone. In a subsequent paper the varying symptoms and pathologic changes of herniation of the lower lumbar intervertebral disks will be considered more specifically in the light of this recognition of demonstrable areas of hypalgesia corresponding to single nerve root dermatomes.

CONCLUSIONS

In the dermatome charts presented by Head and by Foerster anatomic patterns in the lower extremity are difficult to follow because of the finding by these authors of sensory representation of some nerve roots only in the distal region and the confusing extensive overlap recorded by Foerster.

From a study of a large series of cases of herniated intervertebral disk with compression, traction and section of single nerve roots, Foerster's statement that "the amount of overlap of the different dermatomes is so considerable that division of a single root produces no loss of sensibility" is found not to be true for the lower extremity.

The commonest lesion of a single nerve root to be encountered is compression of the first sacral nerve root by herniation of the nucleus pulposus of the fifth lumbar intervertebral disk. The complete syndrome of sensory loss for this nerve root shows a dermatome strip of definite hypalgesia which extends continuously from the little toe to the upper sacral region of the spine, with absence of the ankle jerk.

Loss referable to the fifth lumbar nerve root is found as a dermatome strip of hypalgesia which extends continuously from the middle three toes to the lower lumbar portion of the spine, including an area of hypalgesia on the sole of the foot extending as a wedge from the middle three toes. The ankle or the knee jerk may be reduced.

Loss referable to the fourth lumbar nerve root is found as a dermatome strip of hypalgesia which extends from the great toe to the lumbar portion of the spine, with absence of the knee jerk.

Loss of sensation referable to the third lumbar nerve root is found as a dermatome strip of hypalgesia which extends from the medial surface of the knee to the lumbar portion of the spine, with no loss of reflexes.

Loss referable to the second sacral nerve root is found as a dermatome strip of hypalgesia which extends from the central portion of the sole and the medial surface of the heel to the lower sacral part of the spine. In a spine with six lumbar vertebrae this nerve root overlies the sixth lumbar disk.

On the basis of these observations, a new composite dermatome chart for the lower extremity is presented, representing the areas of definite reduction of pain sensation or hypalgesia due to lesions of single nerve roots. This dermatome chart should be useful for anatomic and neurologic reference.

There is little, or no, overlap of the pain dermatomes plotted in this manner, and the finding of a single dermatome area of hypalgesia in the foot or leg associated with clinical symptoms of unilateral pain in the lower part of the back and sciatic pain accurately localizes the lesion to that nerve root and the herniation to the disk of the segment next above, without the need of intraspinal myelograms.

107 South Seventeeth Street.

Obituaries

CORNELIUS GYSBERT DYKE, M.D.

1900-1943

The untimely death of Cornelius Dyke has robbed the medical profession, particularly the sciences of radiology and neurology, of an outstanding figure. Dr. Dyke was born July 25, 1900, in Orange City, Iowa, of Dutch stock. He grew up and received his college and medical education in his home state.

After receiving the degree of Doctor of Medicine from the State University of Iowa College of Medicine, he joined the United States Navy and took his internship as a lieutenant (jg) at the United States Naval Hospital, Chelsea, Mass. Immediately after his internship he joined the resident staff at the Peter Bent Brigham Hospital, in Boston, in the department of radiology. From the very beginning his radiologic bent was in the direction of the diagnosis and treatment of diseases of the nervous system. This was in 1927, when Dr. Harvey Cushing was still active at the Brigham, and when Dyke's gifted teacher, Dr. Merrill Sosman, was presiding over the richest collection of neuroradiologic material in the world.

While this opportunity colored Dyke's future career, he might have gone on as a general radiologist, with perhaps a special interest in neurologic conditions, but for the fortuitous opening for him that occurred in 1929 on the staff of the radiologic department of the Neurological Institute of New York. This being a large hospital devoted entirely to the care of patients with neurologic diseases, Dyke quickly realized the analogy between his position and that of the radiologists who devote their careers to the diseases of the chest, the gastrointestinal tract and the osseous system. He then and there became a student of neuroanatomy, neurophysiology, neuropathology and clinical neurology. Combining his knowledge of these sciences with a keen eye for detail in the roentgenogram, he became one of the leaders in a highly specialized discipline which may properly be designated as neuroroentgenology.

Following in the footsteps of other great diagnosticians, he never failed to be present at the operating table or the postmortem room to verify his diagnoses or to benefit by his errors. As a consequence, his mistakes became fewer and his diagnostic acumen increased to the point where clinicians began to lean ever more heavily on his aid, and his reputation spread beyond the walls of the institute to neighboring hospitals, then to the rest of the vast city of New York and eventually to the country at large, and to foreign countries as well. His mail was loaded daily with roentgenograms of the skull and spine from far and near for diagnosis. His devotion to his work was such that he never grumbled about these extra burdens, which frequently prolonged his working day beyond measure, and always sent a prompt report, often with some kind word to soften the blow to the vanity of a hard-working colleague in Mississippi or Oregon who had failed to see for himself some obvious lesion.

Official recognition came relatively quickly to Dyke. At the Columbia University College of Physicians and Surgeons he was rapidly advanced from instructor to assistant professor, and then to associate professor. At the Neurological Institute he was made director of the department of radiology in 1939. National societies, both radiologic and neurologic, honored him with membership. Thus, at the time of

his death he was a founder member of the American Board of Radiology, fellow of the American College of Radiology, member of the New York and the American Roentgen Ray Society and first vice president of the last-mentioned society. He was also an associate member of the American Neurological Association and a member of the Harvey Cushing Society, as well as its president in 1940-1941.

Dr. Dyke's contributions to literature were numerous and important, and his books and papers occupy honorable places in the libraries of his colleagues.

His outstanding personal characteristics were his shining honesty and his dislike for nonessentials. It is these qualities which undoubtedly made him so lucid a teacher of his subject.

Outside his work his interests were simple and few. He was devoted to his family and loved music. While he did not regularly attend church, he was nevertheless deeply and sincerely religious.

His death on April 23, 1943 followed several months of illness with acute myelogenous leukemia. His remains were buried in Orange City, Iowa, among the simple graves of his people.

LEO M. DAVIDOFF, M.D.

News and Comment

NOTICE

The Association of Military Surgeons of the United States will hold its fifty-first annual convention in Philadelphia, at the Bellevue-Stratford Hotel, October 21 to 23, inclusive, according to an announcement by officers of the association.

The three day convention will assemble physicians and surgeons from all the current war fronts where United States forces are fighting and from the great base hospitals, where rehabilitation of the wounded is in progress. They will bring with them information on the latest technics of wartime medicine and surgery. Numerous forum lectures, practical demonstrations, moving pictures and teaching panels are planned to present the wealth of data to the convention.

Honorary chairman of the convention this year is Rear Admiral Ross T. McIntire, Surgeon General of the United States Navy. The general chairman is Captain Joseph A. Biello (MC), U.S.N., who is District Medical Officer of the Fourth Naval District.

Abstracts from Current Literature

EDITED BY DR. BERNARD J. ALPERS

Anatomy and Embryology

CORTICAL REPRESENTATION OF THE MACULA LUTEA. TRACY J. PUTNAM and SUMNER LIEBMAN, Arch. Ophth. **28**:415 (Sept.) 1942.

Putnam and Liebman "present a review of the origin and development of the conception of the cortical representation of the macula, with especial reference to the theory of bilateral representation of both halves of the macula. A large number of observations bearing on the subject have been recorded, and more are constantly being reported, often without adequate survey of what has gone before. It appears somewhat unlikely that further new cases will radically alter the broad outlines of the situation. The type of study which seems at present most likely to illuminate the question is a critical and unprejudiced evaluation of the data now available."

A review of the data on the anatomy and physiology of the macula and fovea leads the authors to conclude: "In practically none of the reported cases of 'macular sparing' does the area of preserved central vision correspond with the expected projection of the macula. As defined anatomically in most instances, the preserved central vision subtends only a few degrees. If the entire macula is actually represented in both occipital lobes, there should be approximately as much diminution of visual acuity on the unaffected side as there is retained vision on the hemianopic side after a unilateral lesion destroying a portion of the macular fibers (Wilbrand).

"The problem is not solved by shifting it to the bilateral representation of the fovea centralis, for in some reported instances the spared portion of an incomplete hemianopia is far larger. In other instances still it extends some distance up and down the vertical dividing line, as was pointed out by Wilbrand (1890)."

The authors review the origin of the idea of bilateral central vision, which was first proposed by Bunge in 1884. The decentralist argument arose as a result of the Wilbrand theory of bilateral representation and the von Monakow and Zeeman concept of the representation of the macula in the geniculate body.

"The definitive work of Brouwer and Zeeman, who produced cleancut degenerations in the geniculate bodies of apes by sharply localized lesions of the fovea, proved beyond reasonable doubt that although the macular representation in the geniculate body is extensive, it is nevertheless well localized in the dorsomedial part of the geniculate body. Brouwer and Zeeman stated that their results pointed to a synthesis of the views of the centralists and the decentralists."

Rönne's work seems to prove that macular sparing is merely a special type of hemiamblyopia as a remnant of a lost visual field. "He suggested that when a lesion causes a diminution in function of an entire half of the retina, the peripheral fields fall below the threshold of response to light stimuli. The more densely innervated macular retina, however, having a lower threshold, is still able to preserve some of its sensitivity. He further proposed to account for 'macular' sparing on the basis that the macular cortical center is larger than that of peripheral vision. Hence, there is a greater probability that part of the 'macular' cortex will remain intact even in the presence of large lesions. He suggested that hemiamblyopias are the most common field defects, just as hemipareses are more common than hemiplegias."

Wilbrand's later views, expressed in 1926, renounced the idea of bilateral representation, and he admitted the justification of the criticisms of Lentz and Rönne.

Putnam and Liebman concluded:

"The preponderance of evidence is in favor of an extremely large representation of central vision at the posterior end and in the depths of the calcarine fissure. It is possible that central vision has some representation even at the anterior ends of the fissure and if so doubtless in the portion adjacent to the ventricle.

"There is no satisfactory anatomic evidence of a callosal bundle uniting one geniculate body with the striate cortex of the opposite side, and there is considerable evidence that such a decussating pathway does not exist. The clinical cases on the basis of which such a decussating pathway has been postulated are better explained by other hypotheses.

"Many cases are on record in which a homonymous hemimacular scotoma has existed, but in only 1 of these has the brain been examined in serial sections. The lesion was found at the tip of the occipital lobe. In all cases, the lesion has been traumatic and has presumably injured the optic radiation.

"Lesions of the anterior portion of the striate area on one side produce a contralateral hemianopia with a large remnant of central vision. Lesions of the tip of the occipital pole produce a hemianopia with irregular boundaries, sometimes with only 1 or 2 degrees of central vision. Neither type of central field is actually 'macular.'

"Total or subtotal lesions of one occipital lobe may produce either a complete hemianopia or one with varying traces of central vision.

"The persistence of central vision in cases of a lesion of the occipital lobe may be due to any one of several factors. One is the extensive representation of macular vision, probably occupying over half the area of the visual cortex and possessing a blood supply from three sources.

"Another explanation of the tiny remnants of central vision following extensive lesions is that there is a constant physiologic shift of fixation, so that an object in the hemianopic field but near the fixation point may be brought into view. In some cases, there is evidence of the formation of a new fixation point, within the preserved field.

"Finally, it seems likely that a certain degree of visual perception may be taken over by lower visual centers after occipital lesions, as clearly occurs in monkeys."

An extensive bibliography is appended.

SPAETH, Philadelphia.

IRREGULAR AND MULTIPLE HOMONYMOUS VISUAL FIELD DEFECTS. M. B. BENDER and I. S. WECHSLER, Arch. Ophth. **28**:904 (Nov.) 1942.

The localizing value of field defects is an established neuro-ophthalmologic fact.

"Homonymous central or paracentral scotomas have been reported from time to time, but multiple scotomas in homonymous fields are extremely rare. In reviewing the literature, Wilbrand and Saenger cited several isolated instances of irregular and multiple field defects occurring in patients with gunshot wounds of the occipital lobe cortex and the adjacent optic radiations. In a large series of cases of tumors involving the geniculocalcarine pathways in which careful tangent screen and perimetric studies were made, isolated discrete scotomas were never found."

The authors report 3 cases of multiple scotoma studied over a long period, which permit the drawing of significant conclusions.

"Field defects obviously can occur as a result of a lesion anywhere in the course of the visual pathways, from the retina through the optic nerve, chiasma, tract, geniculate ganglion and optic radiation to the occipital cortex. The absence of retinal changes and optic nerve atrophy at once excludes a lesion in the optic pathways to the geniculate bodies. The preservation of the light reflex and the absence of optic nerve atrophy eliminate a chiasmal lesion. Acute bilateral hemianopic field defects due to a chiasma lesion are usually incongruent, involve chiefly the temporal quadrants and are rarely, if ever, homonymous. The homonymous hemianopias in the authors' cases were due to lesions either in the optic radiation or in the calcarine cortex. . . . A number of the clinical facts point to the cortex as the more probable localization.

"Bilateral lesions are not likely suddenly to affect the radiation, while such lesions are known to occur in the cortex. . . . Because of the compactness of the fibers in the radiations, a lesion there is apt to result in a homonymous field defect. On the other hand, bilateral homonymous hemianopia can and does occur as a result of a lesion in the occipital cortex. Sufficient recovery may rapidly ensue in the case of a cortical lesion, and the wide area covered by the visual cortex permits of irregular involvement and the occurrence of numerous scotomas in the homonymous fields. Finally, there is the additional evidence, . . . in the first 2 cases, of cortical involvement other than the occipital. The visual hallucinations and disturbances in spatial orientation obviously reflected lesions of the cortex and not of fiber structures.

"There remains the question whether a lesion is in the lateral occipital cortex or in the calcarine area. . . . Object agnosia, defective visual memory and disturbances of spatial orientation point to the outer surface of the occipital lobe, but the visual field defects speak of the calcarine cortex or the terminal portion of the optic radiation. This is particularly true of the upper field defects, altitudinal hemianopia, which generally reflect bilateral involvement of the lower lips of the calcarine cortex. It may well be that in the case in which there was spatial disorientation the external surface was also involved, either by an extensive lesion or by edema, which receded sufficiently to bring about partial recovery and left behind only the multiple scotomas in the homonymous fields due to the residual lesion in the striate cortex."

SPAETH, Philadelphia.

THE MOTONEURONS OF THE SPINAL CORD OF THE FROG. MAURICE L. SILVER, J. Comp. Neurol. **77**:1 (Aug.) 1942.

Silver studied sixteen series of the entire central nervous system of the frog, cut in three planes and stained to show myelin, Nissl substance or fibers. In addition, many partial series, prepared with various stains, were available. The position of every motor neuron was plotted and the total number counted. On the basis of the charting of the motor neurons, microelectrodes were inserted into the cell columns at various levels, and localized electrical stimulation was applied. The medial column of motoneurons was found to innervate the axial musculature, and the lateral column, the limb musculature. Synaptic fields were found at the periphery of the spinal cord, at the margin between the gray and the white substance and at the level of the perikarya.

THE GLIAL ELEMENTS OF THE SPINAL CORD OF THE FROG. MAURICE L. SILVER, ibid. **77**:41 (Aug.) 1942.

Using the same material as that in the preceding study, Silver described the glial elements in the spinal cord of the frog. The glial cells have the same fundamental form and relations as the astroglia and microglia cells of mammals. The glial elements outnumbered the neuronal elements 3 to 1.

FRASER, Philadelphia.

Physiology and Biochemistry

SOME MECHANISMS CONTROLLING LOCOMOTOR ACTIVITY IN THE CRAYFISH. W. SCHALLEK, J. Exper. Zool. **91**:155 (Nov.) 1942.

The normal cycle of night activity continues in the crayfish Cambarus virilis even if the animal is kept in constant darkness. The animal becomes continually active if the eye stalks are removed. Injection of sinus gland extract does not affect the activity of animals without eye stalks. In normal animals section of the optic nerve causes increased activity, comparable to that following removal of the eye stalks. The activity of the crayfish appears to be inhibited during the quiet phase by the action of fibers contained in the optic nerve.

Normal animals exposed to four hours of alternate light and dark are quiet in the light and active in the dark, although traces of the underlying twenty-four hour rhythm remain. Stimulation of the caudal photoreceptor makes animals without eye stalks more active in the light than in the dark.

REID, Boston.

THE EFFECT OF BILATERAL OPTIC ENUCLEATION ON THE VOLUNTARY MUSCULAR ACTIVITY OF THE ALBINO RAT. LUDVIG G. BROWMAN, J. Exper. Zool. **91**:331 (Dec.) 1942.

The voluntary muscular activity, as recorded by work-registering cages, of 64 normal-eyed albino rats and 40 rats with both eyes removed was studied under four kinds of light conditions. The animals were derived from a 16 generation brother-sister-inbred strain. The peak of voluntary muscular activity occurs between 2½ to 3½ months of age, after which there is a gradual decline. Normal females are more active than normal males except in constant dark. Blinded males and females are more active than normal males and females except in constant dark.

In continuous light, normal rats have a notable inhibition of spontaneous activity, and the differences between their activity and that of blinded rats is great. If the eyes of adult rats in constant light are removed, there is an immediate increase in activity.

The differences in activity of blinded and of normal rats, as well as the normal sex difference, are obliterated by an environment of constant dark. If the eyes of rats in constant dark are removed, there is no noteworthy alteration in the average daily activity.

Rats kept on a sixteen hour day schedule had three periods of dark in forty-eight hours. The average activity per forty-eight hours was not significantly greater than that of animals kept on the twenty-four hour day schedule (two periods of dark). The absence of light, rather than the frequency with which light is removed, seems to determine the greater activity of the rat in the dark.

A comparison of the activity records of normal rats with those of blinded animals within each series results in three groupings: Normal animals showed (*a*) 48 per cent as much activity as blinded animals in continuous light; (*b*) 72 per cent as much activity in the normal colony and 79 per cent as much in the artificial 16 hour day, and (*c*) 99 per cent as much activity in constant dark.

The blinded rats are less active at warmer temperatures and more active at cooler temperatures. In rats with normal eyes light has a greater influence on activity than does temperature.

The inhibiting effect of visible light on voluntary muscular activity appears to depend on an intact optic mechanism.

REID, Boston.

THE SIGN OF BABINSKI IN MACACA MULATTA. FRANCIS M. FORSTER and JAMES B. CAMPBELL, J. Nerv. & Ment. Dis. **96**:493 (Nov.) 1942.

Forster and Campbell studied the results of plantar stimulation in intact macaques and in macaques after various surgical procedures. In normal animals a flexor response was uniformly obtained. After hemisection of the thoracic portion of the cord no movement of the ipsilateral great toe was produced by plantar stimulation, and when areas 4 and 6 of the same side were ablated in the same animals, the plantar responses were unchanged. Bilateral ablation of areas 4 and 6 had no effect on the normal plantar reflexes, and the additional ablation of areas 3 and 2 produced no alteration. Definite abduction and extension of the hallux, with fanning of the digits in 1 case, was associated with a large contralateral postoperative hematoma. The addition of section of the corpus callosum to hemisection of the cord and unilateral ablation of areas 4 and 6 resulted in an evanescent and inconsistent extensor response on the side opposite the ablations. Hemidecortication was shown to have no effect on the plantar reflexes. Ligation of the anterior cerebral artery at various sites produced a positive Babinski sign, which varied in laterality.

The authors conclude that the Babinski sign in the macaque can be elicited by extensive, bilateral cortical lesions and that apparently no specific cortical area is responsible for its production. Thus the macaque, although possessing a higher degree of encephalization than had previously been suspected, represents a stage in which area 4 has not yet assumed definite control over the Babinski sign. The projection fibers in the cord may be in the ventral column. The transcortical connections, the existence of which is inferred from the bilaterality of the lesions necessary to produce the sign, probably reside in the corpus callosum and the anterior and posterior commissures.

CHODOFF, Langley Field.

SOME FACTORS INFLUENCING THE INTOXICATING EFFECT OF ALCOHOLIC BEVERAGES. HENRY W. NEWMAN and MASON ABRAMSON, Quart. J. Stud. on Alcohol **3**:351 (Dec.) 1942.

Newman and Abramson found that wine with moderate sugar content when administered in divided doses to a fasting subject produces less intoxication than do distilled liquors which give the same concentration of alcohol in the blood. The distilled liquors tested do not vary as to the degree of intoxication produced at a given alcohol concentration of the blood. A large single dose of distilled liquor, because of the rapid absorption, will produce a greater degree of intoxication than will an equal dose of alcohol contained in wine.

The rate of metabolism of the alcohol contained in the various beverages does not differ from that of grain alcohol.

The onset of intoxication during the gradual rise in the alcohol concentration of the blood is not gradual, but begins abruptly when a critical level of alcohol concentration, characteristic of the subject is reached. Above this level, small increases in alcohol concentration in the blood produce marked enhancement of intoxication.

LESKO, Bridgeport, Conn.

THE INFLUENCE OF INTRAVENOUSLY ADMINISTERED ALCOHOL ON THE EMPTYING TIME OF THE STOMACH. LEON A. GREENBERG, GIORGIO LOLLI and MIRIAM RUBIN, Quart. J. Stud. on Alcohol **3**:371 (Dec.) 1942.

The authors raise the question whether the effect of alcohol on the emptying time of the stomach originates locally, from irritation of the stomach, or centrally, from absorption of alcohol or through both mechanisms.

Alcohol was administered to rats by intravenous injection with both dextrose and alcohol as test meals. The experiments demonstrated that even after three hours the stomachs of the experimental animals were distended and full, in contrast to the stomachs of control rats, which were flaccid and contained only small amounts of fluid. The inhibition of gastric emptying observed in these experiments must be ascribed to a central effect of alcohol.

LESKO, Bridgeport, Conn.

ALLEGED DIMINUTION OF WORKING CAPACITY FOLLOWING ACTIVITY OF ANTAGONISTIC MUSCLES. K. WOLF, Arch. f. d. ges. Physiol. **244**:406, 1941.

Clementi observed that the muscles of the jaw, the middle finger and the hand show no diminution of their working capacity if the work is preceded by activity of the antagonistic muscles. He explained this phenomenon as resulting from reciprocal inhibition of the centers of the antagonistic muscles. Reexamination of the muscles of the hand and foot of 5 normal subjects showed that this phenomenon appears only when the activity of the antagonists is associated with contraction of the agonists, so that resting of the agonists is prevented. It was observed, for example, on rhythmic flexion and extension of the hand, the hand grasping a peg, so that contraction of the flexor muscles was innervated on dorsal flexion of the hand. After this source of error had been eliminated, it was found that contraction of the antagonists (i. e., extensors) improved the subsequent performance of the agonists (i. e., flexors of the hand), apparently owing to improvement of the circulation. Thus Clementi's assumption of an after-effect of reciprocal inhibition of antagonists is not accepted by Wolf. He concludes that movements of a limb preceding its actual work may even delay its fatigue provided that the preceding contractions of the antagonists are not performed against large resistance. If the antagonists (i. e., extensors) work against resistance, their contraction is associated with contraction of the agonists (i. e., flexors), so that fatigue of the agonists is accelerated.

SPIEGEL, Philadelphia.

Psychiatry and Psychopathology

THE HYPOTHALAMUS IN PSYCHIATRY. JULES H. MASSERMAN, Am. J. Psychiat. **98**:633 (March) 1942.

Masserman reviews the experimental and clinical data concerning the role of the hypothalamus in emotion. He points out that the activity produced by electrical stimulation of the hypothalamus is artificial and is related only to the stimulus, whereas the stupor and apathy following destruction of the hypothalamus may not be a primary result but may actually be secondary to the metabolic changes produced by such a lesion. Therefore Masserman concludes that the hypothalamus is neither the source nor the seat of experience of the emotions. He studied a series of cats conditioned to peripheral sensory stimuli. Disruption of established responses by conflictual peripheral stimuli resulted in experimental neuroses. Direct hypothalamic conditioning, however, failed to produce a determinative emotional response. Hypothalamic stimulation could not be used as a conflictual stimulus.

Masserman concludes that clinical reports for the most part have been inconclusive and have represented attempts to find a psychiatric application for conclusions previously demonstrated by animal experimentation.

FORSTER, Boston.

HYPOCHONDRIACAL COMPLAINTS WITH SPECIAL REFERENCE TO PERSONALITY AND ENVIRONMENT. SOLOMON KATZENELBOGEN, Am. J. Psychiat. **98**:815 (May) 1942.

Katzenelbogen studied 51 patients with hypochondriacal complaints but without other notable mental disorder. This study was focused on the complaints, personalities and environmental situations. The patients' complaints were referred to the abdominal organs, the head and the heart, in that order, or they had no localization, such as general weakness, backache and insomnia. Occasional patients presented but one complaint. Among the female patients complaints were more numerous and more colored by affect and were more frequently referred to abdominal organs. No general description of the personality traits of either the male or the female patients was possible. The only personality feature common to all was their peculiar attitude toward health and sickness. A majority of the patients had experienced unfavorable life experiences. These episodes might be considered as directly responsible for the initiation of symptoms, but the fixation and protraction were fostered by the hypochondriacal personality makeup.

FORSTER, Boston.

OBSERVATIONS ON MENTAL PATIENTS AFTER ELECTROSHOCK. HANS LÖWENBACH and EDWARD J. STAINBROOK, Am. J. Psychiat. **98**:828 (May) 1942.

Löwenbach and Stainbrook discuss in detail the clinical phases of recovery following electric shock therapy. The gradual resumption of automatic and voluntary activity following the seizure is carefully described. During this process of recovery the authors studied the patient's handwriting and his responses to Rorschach and Wertheimer gestalt pictures. Early in the course of recovery perceptual laxity was evidenced. This was soon followed by perseveration. A gradual increase in the number of total responses occurred as the patient recovered further

from the seizure. Löwenbach and Stainbrook conclude that rapid recovery from the seizure is not desirable, since psychotherapeutic contact can be better established during the period of confusion and disorientation.

FORSTER, Boston.

POSTURAL REACTIONS TO VESTIBULAR STIMULATION IN SCHIZOPHRENIC AND NORMAL SUBJECTS. ANDRAS ANGYAL and MAX A. SHERMAN, Am. J. Psychiat. **98**:857 (May) 1942.

Angyal and Sherman studied 20 schizophrenic patients and 20 normal control subjects in order to determine the postural reactions to vestibular stimulation. The subject stood in a painted circle, was blindfolded and marked time by stepping up and down in the same place. The degree of deviation for each subject was determined, and then the vestibular mechanism was stimulated by caloric tests. Total angular rotation after stimulation was found to be the resultant of three factors: (*a*) the basal turning tendency; (*b*) the turning tendency due to vestibular stimulation, and (*c*) the compensatory tendencies. Angyal and Sherman found that schizophrenic patients compensate less than normal subjects. Although a definitive explanation of these differences is not at present possible, two probabilities are suggested: (1) The nervous mechanisms of the vestibular reactions are depressed in schizophrenia, or (2) the mechanisms function equally well in schizophrenic and in normal subjects, but the differences occur in the function of the effector organs.

FORSTER, Boston.

SOME FORMS OF EMOTIONAL DISTURBANCES AND THEIR RELATIONSHIP TO SCHIZOPHRENIA. HELENE DEUTSCH, Psychoanalyt. Quart. **11**:301, 1942.

Deutsch points out that there are four types of reactions to disturbances of the emotional life. 1. The person is unaware that such a disturbance exists, but its presence is perceived readily by others and in psychoanalysis. 2. The patient complains of an emotional defect and is upset by the disturbance in his inner experience. These complaints may be transitory, may occur only in connection with specific situations or may be continuous. 3. The patient complains that he feels changed—unreal. 4. The patient complains that the external world is changed and has become unreal. Reaction types 2, 3 and 4 are part of the picture of depersonalization. In this paper Deutsch discusses type 1.

The person with a disturbance of the first type seems normal in his emotional responses, but close acquaintance indicates that he reacts only "as if" he were adequate. He is usually intelligent and gifted, understands intellectual and emotional problems but has no originality. His relationships are intense and bear all the earmarks of friendship, love, sympathy and understanding, but they are all devoid of warmth. His attitude to others is completely passive and plastic. He is suggestible, not with the suggestibility of hysteria but with a passive, automaton-like suggestibility. All aggressive tendencies are masked by this passivity. In all cases there is a profound disturbance of sublimation as the result of a failure to synthesize infantile identifications into one person. This disturbance of sublimation results in an imperfect, one-sided intellectual sublimation of ideal strivings. The person seeks external reality in order to avoid anxiety-laden fantasies. Such a person does not repress, but has lost, his object cathexes and his apparently normal relationships are imitative of their environment.

The cause of the condition is associated with (1) a devaluation of objects; (2) an insufficient stimulus for the sublimation of emotional reactions, because they were treated with too little or too much tenderness, or (3) failure to form enough defense mechanisms because the child was treated too harshly or too indulgently.

The condition must be differentiated from the following conditions: 1. Hysteria. In hysteria there are powerful libidinal object cathexes. The hysterical person remains free from the anxiety caused by these cathexes by repressing affect. In such persons the early deficiency in the development of affect reduces the inner conflict. 2. Blocking of affect through repression. In this disorder there is not the simulated affective experience exhibited by the "as if" personality. 3. Psychosis. In a psychosis the faculty for testing reality is lost; not so with the disturbance under discussion. The patient with a manic-depressive psychosis introjects the whole conflict situation. The patient with emotional disturbance keeps the conflict situation external, and the conflict is acted out. It is the author's impression that all schizophrenic persons pass through the "as if" stage. 4. The pseudoaffectivity seen in puberty. This may be the normal expression of a condition of which the "as if" stage is the pathologic form.

Psychoanalysis seldom cures patients with this disorder, but can improve them greatly. The analyst has a unique opportunity to learn a great deal about ego psychology.

PEARSON, Philadelphia.

DEFENSE REACTIONS IN ANXIETY STATES OF CENTRAL ORIGIN. J. KASANIN, Psychoanalyt. Quart. **11**:493, 1942.

Anxiety consists of three parts: a quality of psychic discomfort; physiologic concomitants involving both the sympathetic and the parasympathetic system, and a psychic perception of these sensations. Anxiety is a warning signal which results in the initiation of activity by the ego in order to protect itself by removing the source of the danger. Kasanin believes the anxiety seen in the neuroses is not primary, but one that has been secondarily modified by the ego. He cites 2 cases. In the first case, a young woman showed all the signs of anxiety without the subjective perception when she was slightly aroused sexually. Removal of a small adrenal tumor resulted in disappearance of the symptoms. In the second case, a boy aged 11 years presented the clinical picture of depression without its subjective perception. He had a teratoma of the pineal gland. He showed two defense mechanisms against allowing himself to feel the unbearable anxiety aroused by his approaching death: He denied the reality of his illness, and he projected his inability to eat onto his mother and sister, saying that they could not eat; then, by identifying himself with them, knowing they were capable of eating, he denied that he was in danger of death.

Kasanin thinks that when the organs which participate in the physiology of fear and anxiety, the adrenals and the hypothalamus, are involved in a disease process, a primary type of anxiety is evoked, which differs from ordinary anxiety in the character of the subjective content.

PEARSON, Philadelphia.

Diseases of the Brain

ORTHOSTATIC HYPOTENSION ACCOMPANYING THE TABETIC FORM OF DEMENTIA PARALYTICA. HAL E. FREEMAN and JAMES E. ROBERTSON, Arch. Dermat. & Syph. **46**:796 (Dec.) 1942.

Freeman and Robertson report a case of postural hypotension associated with the tabetic form of dementia paralytica. The condition has been described in tabes but not in this disease. Malarial treatment was well tolerated.

ALPERS, Philadelphia.

FOSTER KENNEDY SYNDROME ASSOCIATED WITH NON-NEOPLASTIC INTRACRANIAL CONDITIONS. H. EDWARD YASKIN and N. S. SCHLEZINGER, Arch. Ophth. **28**:704 (Oct.) 1942.

This syndrome, first discussed in 1909 by Leslie Paton, was further considered and amplified in 1911 by Kennedy. The symptom complex comprises unilateral atrophy of the optic nerve with concomitant contralateral papilledema. The variations in the fields of vision are cited by Yaskin and Schlezinger as central scotoma with primary atrophy.

The authors report 2 cases in which the clinical features of the syndrome were presented; in neither instance was the condition the result of an expanding lesion of the basofrontal region or of the anterior fossa. "The neuroretinal disturbances were a result of compression of the optic nerves by sclerotic vessels, proved in the first case and probable in the second case. The presence of a binasal field defect in association with the Foster Kennedy syndrome necessitates consideration of the diagnostic possibility of sclerotic internal carotid or anterior cerebral arteries. Cases reported in the literature also indicate that the syndrome may be caused by an intracranial neoplasm which is not situated in the anterior fossa. In all doubtful cases of a neoplasm of the anterior fossa in which a Foster Kennedy syndrome exists, one should resort to arteriography or cerebral air studies before recommending craniotomy."

SPAETH, Philadelphia.

HEMORRHAGES INTO THE LATERAL BASAL GANGLIONIC REGION. CYRIL B. COURVILLE and ARNOLD P. FRIEDMAN, Bull. Los Angeles Neurol. Soc. **7**:137 (Sept.) 1942.

The authors report 31 cases of recent and old hemorrhage into the lateral ganglionic region. It is well known that hemorrhage in the medial portion of the ganglionic area of the brain results in death, but it is not recognized that nonfatal hemorrhage may occur in the lateral ganglionic area. The lateral ganglionic region is made up of the putamen, the external capsule, the claustrum and the regional (insular) subcortical white substance, sometimes designated as the capsula extrema. The lateral ganglionic region is therefore to be distinguished from the medial ganglionic region. The division between the two regions falls along the septum which separates the globus pallidus from the putamen.

A large hemorrhage in the lateral ganglionic area of the brain results in death in the majority of cases. A small hemorrhage does not result in the profound or prolonged coma that is present with effusion into the medial ganglionic region. The clinical picture of hemorrhage in this area is one involving both the motor and sensory systems and the faculty of speech.

LESKO, Bridgeport, Conn.

MASSIVE HEMORRHAGE INTO THE CAVUM SEPTI PELLUCIDI. CLARENCE W. OLSEN, Bull. Los Angeles Neurol. Soc. **7**:152 (Sept.) 1942.

Olsen reports a case of hemorrhage into the septum pellucidum resulting from bleeding of the anterior communicating artery. From clinical observation in cases with cyst, tumor or hemorrhage in the region of the septum pellucidum, several features of a frontal, subcallosal, interhemispheric syndrome have been observed, namely, dementia disorientation, confusion and emotional instability associated with papilledema.

LESKO, Bridgeport, Conn.

THE PRINCIPLE OF PRIMARY AND ASSOCIATED DISTURBANCES OF THE HIGHER CORTICAL FUNCTIONS AS APPLIED TO TEMPORAL LOBE LESIONS. HARRY A. TEITELBAUM, J. Nerv. & Ment. Dis. **96**:261 (Sept.) 1942.

Teitelbaum has evolved a method of investigating primary and associated disturbances of the higher cortical language functions by the use of posthypnotic suggestion. By primary disturbances he means those directly resulting from the implanted suggestion, while the disturbances not derived directly from the formula recited to the subject he considers as associated. The author attempts to use this principle in explaining some of the puzzling discrepancies in cases of aphasia. Thus, in cases of amnesia, in which the patient can complete a song when given the first few notes, the condition is considered as an associated disturbance of higher cortical function in which the physiologic substrate of the psychologic act is not destroyed, but only damaged, and is subject to improvement. The discrepancies which exist between the clinical symptoms and the postmortem observations in cases of aphasia are explained on the grounds that associated disturbances require no somatic lesions. In illustration of this, reference is made to 5 cases reported by Henschen, with lesions confined to the first and second temporal gyri, in some of which the word deafness considered characteristic of lesions of the temporal lobe was complicated by such manifestations as word blindness, aphemia, agraphia and jargon aphasia. Teitelbaum considers the latter to be associated disturbances with impaired psychologic mechanisms but with intact organic association pathways. Further analysis of Henschen's data reveals cases of word deafness in which the superior temporal gyri were intact. The author believes that such disorders may be accounted for by interference with other components of a neural association pathway necessary for understanding words. It is because lesions impinge on the essential parts of neural association pathways for other functions that disturbances in word understanding arise.

Another observation of Henschen's which requires explanation is the existence of various disturbances in higher cortical functions in all sorts of combinations. The author believes that this, also, can be accounted for on the basis of the principle of primary and associated disturbances.

CHODOFF, Langley Field, Va.

CERVICAL RIB AND HYPERHIDROSIS. E. D. TELFORD, Brit. M. J. **2**:96 (July 25) 1942.

The cervical rib syndrome is one of muscular weakness, pain and possible vasospastic phenomena. Hyperhidrosis is uncommon. Telford explains the excessive perspiration on the basis of irritation by the cervical rib of the sudomotor sympathetic fibers which run in the lowest trunk of the brachial plexus. Of 92 patients with cervical rib on whom Telford operated, 2 complained chiefly of profuse hyperhidrosis of the affected hand. In each case prompt and complete recovery followed surgical removal of the rib.

ECHOLS, New Orleans.

HYPEROSTOSIS FRONTALIS INTERNA. C. T. ANDREWS, Brit. M. J. **2**: 185 (Aug. 15) 1942.

Andrews states that hyperostosis frontalis interna had often been noted post mortem but that little interest was taken in the condition until Stewart and Morel noted the association of symptoms suggestive of hypothalamic disturbances. The syndrome is characterized by disordered fat metabolism, an increased dextrose tolerance, polyuria and polydipsia, disorders of sleep, progressive dementia and possible palsies of the cranial nerves. The condition involves preponderantly the female sex, the majority of cases occurring during the menopause. Roentgenograms usually show the hyperostosis as an irregular thickening of the vertical part of the frontal bone, the region of attachment of the falx being free. The surface may be irregular, with numerous spikes, or smooth and nodular. The outer table of the skull is never involved. Andrews describes the case of a woman aged 45, characterized by narcoleptic attacks, which were controlled by amphetamine, increased dextrose tolerance, sudden gain in weight, restricted fields of vision and nerve deafness. The importance of the syndrome lies in its differentiation from conditions requiring active medical or surgical intervention.

ECHOLS, New Orleans.

Society Transactions

NEW YORK ACADEMY OF MEDICINE, SECTION OF NEUROLOGY AND PSYCHIATRY, AND NEW YORK NEUROLOGICAL SOCIETY

CHARLES DAVISON, M.D., *Chairman Section of Neurology and Psychiatry, in the Chair*

Joint Meeting, Nov. 10, 1942

Selective Use of Electric Shock Therapy as an Adjuvant to Psychotherapy. DR. HERMAN SELINSKY (read by DR. WILLIAM A. HORWITZ).

The indiscriminate use of electric shock therapy is deplored, and it is urged that this therapeutic measure always be supplemented with adequate psychotherapy. Certain psychiatrists have raised objections to this form of therapy for mental disorders, principally to the effect that it is cruel and crude. In answer, it may be pointed out that the benefit obtained from such treatment is now a matter of undisputed clinical record. Electric shock therapy has been particularly valuable in the psychiatric treatment of ambulatory patients who are still able to earn a livelihood, patients who might hitherto have been confined to a mental disease hospital.

Several case histories are given to illustrate a psychiatric problem which is disconcerting to manage: that of the patient who does not respond to competent psychotherapy or one who is close to being psychotic. It is recommended that electric shock therapy be considered for such selected patients; response to further psychotherapy may be gratifying.

The theoretic considerations for evaluation of psychophysiologic processes involved in the production of improvement are briefly discussed. So, also, are the impairment of memory and the psychomotor manifestations in the seizure pattern.

DISCUSSION

DR. LOTHAR KALINOWSKY: Although Dr. Selinsky himself was cautions in his conclusions, I should like to warn against possible misunderstandings regarding the effect of shock therapy in general. The author applied electric shock for the purpose of making patients more accessible to psychotherapy. I, personally, have never used the method for this purpose. However valuable the shock therapy might be, it is something quite different from the usual treatment of the major psychoses. Depressed and schizophrenic patients improve with surprising constancy after the third or the fourth treatment. This cannot be explained on the basis of a merely psychotherapeutic approach. Comparison of the results with my patients in the New York State Psychiatric Institute, who received the treatment voluntarily, and the results with the institutional patients at the Pilgrim State Hospital reveals that the latter group, who usually have a more severe break with reality, benefited most from the shock treatment, although they received no psychotherapy. Psychologic theories of the effect of this and of other shock treatments are interesting but have no foundation in clinical facts. Dr. Selinsky found patients more accessible to psychotherapy when under electric shock. This might be due either to the slight organic confusion or to improvement with electric shock, so that the patient was in better rapport with his environment, and so with the psychotherapeutist. During the usual routine treatment of three convulsions a week, the psychotherapeutist is generally unable, owing to the patient's impaired memory, to treat him at all. My associates and I have concentrated recently on the effect of electric shock therapy of the psychoneuroses. So far results are not encouraging, with some exceptions, the most important of which are noted with the psychoneurotic depressions. It is because I am convinced of the great value of electric shock therapy for the affective disorders, as well as for acute schizophrenia, that I am cautious in its recommendation for disorders for which its value is not yet established.

DR. R. B. MCGRAW: Dr. Selinsky suggested that part of my discussion be devoted to the feasibility of the use of electric shock in office practice under proper conditions. In a series of 40 cases which I have encountered in office practice there have been only 3 in which minor

injuries occurred, and those were of torn muscles. These injuries were in different locations, but all in the upper part of the body. The proper securing of the patient, either by attendants or by properly applied restraint, without absolute rigidity, is, I think, essential. The after-care should be strict and requires the help of a nurse. Under such precautions I feel that this procedure can be carried out safely in office practice.

I do not believe that the threat of death is important, and I have not been quite so impressed as has Dr. Selinsky with the importance of the release of aggression in all cases, though I am sure it is important in some. I am rather more inclined, as Dr. Kalinowsky is, to the organic or physiologic interpretation of the effect of electric shock. And that makes a difference with respect to psychotherapy. Dr. Kalinowsky mentioned that physicians at the Psychiatric Institute had difficulty in conducting psychotherapy. There is some temptation to think that suggestion could be used with this treatment, but it cannot. The patient does not remember what he is told, and suggestion has no effect, except in a general way. An individual suggestion at a treatment session is of no value at all. The unconsciousness associated with shock has nothing to do with suggestibility, and this is true also of the period of confusion after the shock. The confused state is not like that before or after anesthesia, for example, when suggestion is of value. After the treatment one has to tell the patient many times where he is and how and what the treatment has been about, even to remind him that he has had the treatment. It is obvious, therefore, that no valuable suggestion or directive or reeducative process can be introduced at this time.

As to why electric shock is curative, I believe that the disturbance of newer habit patterns permits older ones to show themselves and that if these older patterns are reasonably healthy the treatment is successful. Another possible explanation is that in some way the electric shock physiologically speeds up the process of recovery.

It would be interesting to see whether a person who had acquired a new language would, if given electric shock treatment, lose the ability to use that language. I have frequently seen patients use their older language in the confused state following electric shock, but I do not know of any instance in which ability to use a new language has been lost. In some patients there are, of course, a release, which has been spoken of, and statements and actions which indicate that the deeper layers are revealed and that the problems and conflicts are therefore understood, and this information can be utilized in psychotherapy.

Dr. Gustav Bychowski: I have been working for some time on the problem of combining psychoanalytic procedures and shock therapy, and I was interested in listening to this paper. I fully share the conviction of Dr. Selinsky that the combination is extremely valuable, and it seems to me that in many cases only shock therapy, especially electric shock therapy, makes possible the application of psychotherapy, which otherwise would be unsuccessful. It can be observed that in cases of complete detachment from reality, in which no transference is possible, one is able to affect the blockage after a few electric shocks.

Careful clinical observation confirms the opinion that the action is psychophysiologic. Neurologic reflexes, which are obvious in the pathologic condition, are removed as an effect of the treatment, and, in perfect parallelism with this neurologic effect, the psychologic block is also broken up. I do not agree with Dr. Selinsky and some other investigators that the fear of death is the main psychologic factor. A great many patients who have been under my observation never had this fear, or it did not seem to play an important role after the patient had become familiar with the treatment. From the psychophysiologic and the psychoanalytic point of view, the combination of shock therapy and psychoanalysis seems important and valuable because it enables one to study carefully the structure of repression in patients who are otherwise completely inaccessible. One can observe clearly how the retrograde postshock amnesia carries with it the repressed material, and how during the recovery this material comes up. This repressed material, which was completely inaccessible, then becomes accessible, and from then on belongs to the system of the preconscious; thence it can be brought up by simple psychologic procedures, which are similar to those used in any analytic therapy.

Dr. Foster Kennedy: This discussion is based on an error. True, only a verbal error, but one handed down for two thousand years, that is, that there is a thing called the mind and a thing called the body—an ecclesiastical error, which has lamed man's thinking since the time of Origen and Celsus. There is no difference between mind and body, any more than there is between a root and the flower of the root. Psychiatrists usually feel themselves too remote to investigate the soma, and the general physician does not often think of investigating the mind. I believe (it is fantastic, perhaps) that it is only an evolutionary accident that we human beings can see each other's bodies and only deduce each other's minds. It might have happened that we could see each other's minds clearly but must deduce each other's faces, which God forbid! (He did!) Each one of us is an arrangement, a geometric

formula, of electrical particles of energy, and we have produced special senses by which, in a certain aspect and in a limited plane, we can perceive each other. This arrangement of forces which we call ourselves, fragments arranged to make different personalities, is implanted by the "distilling" apparatus, the nervous system, which produces thought and self awareness, compassion and aspiration out of the primitive consciousness of every cell—again, one must use the word "cell," instead of the word "purpose," which every cell contains. One might as well say "purpose" as "cell"; only one can see cells, and one has to think about purpose, and man does not like to think. Psychoses are errors in the flow of energy in the pressor and depressor systems. They are "organic," if I may use the word "organic," and I have to use the vocabulary of dualism because there is no other. The psychoses are "organic," and electric shock, in some way not yet understood, tends to restore the balance. It instantly abolishes consciousness for the time being. It restores earlier phylogenetic patterns; I think Dr. Kalinowsky believes that if such pattern is healthy the patient becomes healthy. The mechanism of this treatment is not yet understood, but it is concerned with the unstable equilibrium of opposed forces by which man lives. It is a physical therapy. That is not to say that the restored patient, restored in his organic balance, cannot be made better by education. Of course he can, just as every one can. "Educo" does not mean "to push in"; it means "to lead out"—to lead out of the patient things that are there, that he has, that the electric shock has partly restored to him.

DR. WILLIAM A. HORWITZ: I am not going to answer the questions addressed to Dr. Selinsky in his absence, especially as I differ in slight ways from him. From Dr. Kalinowsky's remarks, one might get a more severe impression of the inadequacy or worthlessness of psychotherapy as an aid in the treatment of certain types of illnesses than Dr. Selinsky meant to convey. As he pointed out, there are patients who will get well without psychotherapy, but that is not the type referred to here. Certainly, while the patient is having two or three electric shock treatments a week, it is almost impossible to give him psychotherapy because of temporary loss of memory. The patient, however, to whom Dr. Selinsky refers is one who has been treated over a long period and who presents certain resistances which are in the way of his understanding of his problems. With such a patient it is possible to use a psychotherapeutic approach, especially when he is inhibited in his affective responses, and thus to give him better insight and understanding into some of the difficulties that are presented.

Sarcomatosis of the Leptomeninges: Report of a Case. CAPT. M. P. ROSENBLUM, Medical Corps, Army of the United States, and DR. LEWIS D. STEVENSON.

D. C., a woman aged 33, was admitted to the Psychiatric Division of Bellevue Hospital in August 1938, with paralysis of both legs, blindness and loss of control of the sphincters.

In December 1937, in St. Vincent's Hospital, she had had a laminectomy, involving the eleventh dorsal to the third lumbar vertebrae, for a possible tumor of the cord. No tumor was encountered, but the diagnosis of an inflammatory disease of the meninges was made. In July 1938 she became blind. On admission to Bellevue Hospital she presented a transverse lesion of the cord at the fourth dorsal segment, with changes in the fundi (papilledema), bilateral weakness of the sixth nerve and involvement of the right twelfth nerve. There was some rigidity of the neck. Lumbar puncture was done with difficulty, and a small amount of yellow spinal fluid was obtained.

Autopsy revealed extensive sarcomatosis of the leptomeninges, with invasion of the dorsal portion of the cord and extension of the sarcoma upward to cover a good deal of the brain stem, the optic nerves and the cerebral hemispheres.

DISCUSSION

DR. JOSEPH H. GLOBUS: I accept fully the case as one of primary sarcomatosis of the meninges. I also agree with the authors that the problem of primary sarcomatosis of the brain deserves greater attention than it is given.

As far back as 1934, in presenting the subject of primary meningeal tumors, I emphasized that meningeal tumors, being of mesodermal origin, behave as do any other tumors of such derivation, since practically all structures which are derived from the mesoderm are capable of undergoing malignant transformation. Since a great many tumors derived primarily from the mesoderm acquire a sarcomatous character, meningioma, for the same reason, frequently presents the structure of sarcoma. I indicated then that primary sarcoma may occur within the substance of the brain and may be traced to the meningeal covering, since the meningeal covering, particularly the pia, sends its extensions into the substance of the brain by way of

the blood vessels. Therefore the presence of primary sarcoma in the substance of the brain should not be regarded as unusual. Certainly, a sarcomatous tumor which has spread through the meninges of the brain and spinal cord should not be regarded as exceptional.

I have under study a group of 13 cases of primary meningeal tumor; of these, 10 are being prepared for publication. They, like the present case, are undoubtedly instances of primary sarcomatosis of the meninges. They present practically all the features described by Dr. Stevenson.

How does one account for the fact that this tumor not only involved the leptomeninges but spread into the dura and beyond it? The explanation is in full accord with the diagnosis of sarcoma. It need but be realized that the meninges are derived from the mesodermal primordium, which at one time constituted the material from which the covering of the nervous system developed. This primordium gives rise to the pia, the arachnoid and the dura; even the endosteum is traceable to this primitive material, the so-called skeletomedullary intertissue. If this primitive tissue is the source of all the covering membranes, then a tumor of this derivation might occur not only over the surface of the brain, but within the brain and the subdural and epidural spaces.

Dr. Stevenson let me see some of his slides in this case. In one section I was able to see cells containing melanin pigment, chromatophores filled with this pigment. The pia normally contains chromatophores; this, again, is proof that this tumor is derived from the mesodermal primordium, which also contains the material capable of differentiating into chromatophores.

Should this idea, which I suggested some years ago, namely, that there is a common mass of material from which all the coverings of the nervous system have developed, be accepted (and it is not entirely accepted as yet), and that this material is mesodermal (and this is not fully agreed on), the question of primary sarcomatosis of the brain is clarified, and the frequent occurrence of that condition may be accepted without much difficulty. In my material the number of primary sarcomas of the nervous system is approximately 13 per cent of the total number of tumors of the meninges.

Now that all have agreed that the tumor is sarcomatous, that its derivation is mesodermal, and particularly from the pial component, the present case may be accepted as an excellent illustration of this type of tumor.

Limitations of Psychoanalytic Treatment. DR. HERMAN NUNBERG.

An attempt is made to show why psychoanalytic treatment fails in certain cases. A parallel is drawn between the failures of psychoanalytic treatment and the failures of treatment in other branches of medicine. The failures of psychoanalysis are caused by the so-called resistances. They counteract the attempts of the psychoanalyst to help the patient overcome his neurosis. The resistances are numerous; some are more important for the treatment and some less. In a neurosis the ego is relatively weak; it cannot cope with the urges of the instincts and the pressure of the conscience. The analysis tries to strengthen the ego, mainly by encouraging it to face the supposed dangers of the instincts and the conscience. Expressed in most general terms, the analytic treatment fails in cases in which it does not succeed in mobilizing enough psychic energy to enable the ego to cope with the dangers of its inner repressed life.

DISCUSSION

DR. A. A. BRILL: I agree with everything that Dr. Nunberg has said, as I am sure do all the analysts present but I am just as certain that to the physician who is not well versed in theoretic analysis Dr. Nunberg talked Greek. Dr. Nunberg has done a great deal of work on the structure of the ego, and his thesis is woven around that part of the problem. He has given a masterly presentation of some of the difficulties one encounters in analytic therapy. He could not, of course, take a special problem and elaborate fully on it. He covered so much space, however, that it was, in my opinion, somewhat difficult even for an audience of phychoanalysts to follow him.

Psychoanalysis is not a panacea; Freud always maintained this, and it has been stressed by others over and over again. I have always been careful in selecting my patients for analytic therapy. If I did not feel that I could cure a patient, I did not take him. I remembered Freud's statement that it is best to take the patients for analysis whom one feels one can help; the others will drift from office to office, no matter how much effort one makes in their behalf. Let me mention briefly a few cases in my experience in which I felt analysis would not be of benefit.

A single woman, about 38 years of age, was referred to me by a physician for analytic therapy. She had undoubtedly a mixed neurosis; her symptoms were of a hysterical com-

pulsive type, mixed with all sorts of anxieties. She was the eldest of seven children. She was only 19 years of age when her mother died, but she took charge of the home and acted as a mother to the family. She sent them through school and college until they all became independent, and most of them married. A few years after her father died, there was nothing more that she could do. None of her sisters or brothers needed or wanted her; so she became neurotic. Her physician told me that he had prescribed a rest cure, as well as other therapies, and she had benefited from them temporarily, but she was not cured. I saw her twice and then told him that it would be best that he continue to treat her as he had before. I told him that psychoanalytic therapy would not do her any good. It was too late to try to help her adjust to the realities confronting her in life; I felt that her age and general makeup were against any adjustment which psychoanalysis could offer and that I would rather give the time to a more promising case.

A physician who met me at a meeting some time ago said: "About a year ago I wanted to send a woman to you for treatment, but somehow I did not, and now she is all right. If I had sent her to you, you would have gotten the credit; as it is, I get the credit." He added, "And the remarkable thing is that she got well in spite of the fact of a terrible misfortune, her husband's death." As a psychoanalyst, I had a different interpretation of this cure, but there was no use in telling it to my friend.

There are many other patients who are sent for psychoanalysis who cannot be so treated, for environmental reasons. Dr. S., a cardiologist, sent me such a man who thought he had heart trouble. Dr. S. assured me that there was nothing wrong with the man's heart and that the same opinion had been expressed by other cardiologists. The patient consulted me last spring, when I could not take him for regular analytic treatment; so I asked him to return in the beginning of October. In taking his history last May, and again in October, he assured me that there was nothing wrong with his emotional life, that he was a prosperous business man, that he got along well with his wife and that he was pleased with his young son. Nevertheless, he had pain in the region of the heart and other symptoms of a somatopsychic nature. I asked myself: "What is the matter with this fellow? He undoubtedly has hysterical conversion symptoms with anxiety. Yet there is nothing to account for his neurosis." I asked him again about his wife, and he repeated that she was a very efficient and good wife. I recalled that Bleuler and Jung used to say that in taking a history one must always ask oneself, *"Wo steckt die Libido?"* (Where is the libido?), when one is in doubt about the case. I then told him that I should like to talk to his wife. I found her an interesting, aggressive woman, who ruled her husband with an iron hand. Despite his business efficiency, she had a very low opinion of him as a man. I then asked her what she thought of her husband's coming to me, and she said that she considered it degrading, that a man should be able to take care of himself. In brief, she had been married to him for about twenty years and had always considered him a weakling, and she had repeatedly told him so. Yet he kept everything to himself and spoke in high terms of his life with her. On his next visit to me, I asked him what his wife had said about her visit to me, and he said, "She thinks I am an idiot for coming here." Then for the first time he began to unburden himself. I never heard such an outburst of hate and resentment. I could not stop him. The lid having been lifted, everything came out like an eruption. He blamed her for the death of their two children; she had refused to get a doctor when they were sick. One of them had appendicitis, and she not only refused medical aid but forced the child to eat; the appendix ruptured, and the child died. This visit was about a week ago. The man has left his home, but I have no doubt that he will return soon. But all his pain is gone. I feel that little can be done for him by analysis. A man who has been over twenty years under the complete domination of such a wife is, in my opinion, not a promising subject for psychoanalysis. The outburst may help him for a while, but he will probably return to his former behavior.

Dr. Fritz Wittels: I agree with Dr. Brill that this paper can be understood better by one's reading it, which I had the opportunity of doing. I must disagree with Dr. Brill that only an analyst can understand it. Dr. Nunberg explained the limitations of psychoanalytic therapy in language which every psychiatrist should be able to follow. He covered only some of the limitations of the treatment. Let me speak of another which is often overlooked: the limitation involved in the personality of the analyst. It is not only the patient; it is the physician who counts; if the analyst does not know how to behave, what his "bedside manners" must be, he will work in a vacuum.

Dr. Nunberg spoke of the patient's need for punishment, his negative therapeutic reaction, which sometimes becomes an insurmountable obstacle to treatment. Persons feel guilty for

a reason unknown to themselves. Sometimes they know what they have done but do not realize its importance. They feel punishable, and then they cannot stand a physician who is friendly to them. For example, a woman aged 20 had started an affair and had also begun to drink. She did it as a revenge against her widowed father, who she learned had a mistress. But she did not know that there was any connection between the loss of her virginity and her father's affairs. She was confused and depressed. When she came to me I played the wrong tune; I was too friendly and reassuring to a girl who felt guilty and punishable, and she left me after two visits.

I do not mean to say that the psychoanalyst should play a role contrary to his nature. He should not play the strict and stern man if he is by nature inclined to be overtly friendly. He should be completely professional, cool and objective. He should wait with any therapeutic activity until he has a clear insight into the structure of his case.

DR. CLARENCE OBERNDORF: Dr. Nunberg has presented clearly the conflict between the instinctual drives, the ego and the superego. However, the general subject of the limitations of psychoanalytic therapy has not been touched on. The problem with which the analyst is confronted today is this: The patient is chosen for psychoanalysis after a long investigation, anywhere from two weeks to a month, because the analyst feels that the case is suitable; then, after a time, the analysis fails or peters out. This experience falls to every analyst; recently the analysts themselves have been facing the problem more courageously and have learned to trace where the fault lies—whether it is in the intrinsic personal problem, such as Dr. Nunberg has described, or in external circumstances, to which he has given little attention in appraising the final results of analysis, or in the unsuitability of the method to the patient.

I cannot recall a report by a single analyst on his own series of consecutive cases in which he indicates his results according to his own criteria.

Dr. Nunberg mentioned the patient's resistances, and Dr. Wittels carries it a little further, saying that the patient may have infinite capacity to suffer. I might say, too, if the patient has infinite capacity to suffer, the analyst may have a desire to do something for the desire to suffer.

Freud once stated that every analyst should himself be analyzed every five years so that he may see where is own mind is drifting, and whether he has formed habits of interpretation as stubborn as those of his patient's resistance. Few analysts have been willing to submit to this periodic analysis. However, of recent years it has become a custom in psychoanalytic instruction for the novitiate to have his work controlled by an old analyst. It is just as easy for the older analyst to get into habits which are a little too set, a little arbitrary. It would not be a bad idea if every patient who has been under analysis over two years were submitted to a second analyst to find out what the progress has been, what the limitations are, whether the method itself is applicable to the patient's case and whether there is any real hope for an ultimate cure or further benefit.

DR. E. D. FRIEDMAN: Dr. Brill expressed the opinion that Dr. Nunberg's paper would be difficult for those who are not analysts to grasp, but I have seldom heard a clearer or more objective presentation of the value of psychoanalysis. It is good to know that there are psychoanalysts who are not sitting on Olympus, talking down to the rest, who are groveling in the dust of ignorance and who have no understanding of psychoanalysis.

In the forum of the New York Neurological Society the statement has been made that manic-depressive psychosis, paranoia and compulsion neurosis can be cured by psychoanalysis. It is a considerable step forward when one can speak of the limitations and the failures of psychoanalysis. We organicists freely admit we are not capable of curing all neural illness, and so it is cheering to hear that psychoanalysts, also, are not capable of producing a panacea.

It is important to remember that the value of psychoanalysis lies chiefly in the field of the transference neuroses; if the analyst does not accomplish transference, the treatment does not produce results. It was also good to hear Dr. Nunberg say that constitutional factors and rigid character traits may prevent good results with analysis.

Psychoanalysis, in the mind of Dr. Nunberg, has passed from the stage of infantile omnipotence to adolescence—capable of at least some degree of self criticism. Time and truth here, as in other fields of human thought, are allies.

DR. HERMAN NUNBERG: I was ready to take my punishment and not talk any more before this society since Dr. Brill and Dr. Oberndorf thought that my paper was too difficult for such an audience. But now I have changed my mind; I am grateful to Dr. Friedman for having shown that my paper was not obscure and has been well understood by those who are not psychoanalysts.

ILLINOIS PSYCHIATRIC SOCIETY

FRANCIS J. GERTY, M.D., *President, in the Chair*

Regular Meeting, Nov. 5, 1942

Psychiatry in a Critical Period. DR. FRANCIS J. GERTY.

In 1864 S. Weir Mitchell, W. W. Keen and George R. Morehouse published a minor medical classic, "Gunshot Wounds and Other Injuries of the Nerves," in which is reported some of the work of the United States Army Hospital for Diseases of the Nervous System, organized in May 1863 as the first hospital of its kind in the military history of the nation. Except for some brief notes on malingering, nothing seems to have come from this hospital, and not much appears on mental disorders in all the medical literature of the Civil War. The Spanish-American War still reflected but little change in the field of psychiatry, though a few articles on military psychiatry can be discovered. By 1918, during World War I, it was apparent from the number and kind of papers published that psychiatry was in a vastly different state than it had been twenty years earlier. Its contributions had not had much effect on military matters in the prewar period. War and postwar psychiatric problems indicate this. Before this country entered the present war, it was evident that the increased information in the field of psychiatry should be brought to bear before men were inducted into the military service rather than afterward, and that fitness for types of duty of recruits and programs of rehabilitation of rejectees were within the psychiatric purview.

The test of how effectively this knowledge has been applied will come in the future. Psychiatrists may not have had full opportunity to apply what they wished to contribute in the way that they wished, but they have had more opportunity than ever before. Of recent years psychiatry has been a major department in many of the leading medical colleges. The descriptive, classificational and institutional psychiatry, generally one of hopelessness, has been replaced by a mechanism-revealing, experimentally grounded type of research into causes and treatment. One is still somewhat troubled by "schools" and the fervent loyalties of their adherents. Nevertheless, they are all concerned with fundamental matters—technics of treatment that give serious consideration to etiologic factors in emotional and behavior disorders. The seemingly unpromising field of organic psychoses has yielded brilliant results in the treatment of meningoencephalitic syphilis. The graver constitutional psychoses have been vigorously, though empirically, attacked by several types of shock treatment. Enthusiastic proponents and pessimistic critics are still contending as to the merits of these treatments. With regard to the problem in general, it is hoped that the verdict of the future will be that as psychiatrists we have met our present challenge well. It is believed that if we do not meet it well the reason will lie in the deficiencies of our knowledge, rather than in the lack of opportunity to apply the knowledge. Therefore we must work to discover in which ways we can improve our knowledge. As to the functional disturbances, a renewal of psychologic investigations, with the practical view of finding shorter and more effective methods of securing lasting results in treatment, must be our object. The seemingly effective results in the treatment of psychoses by physical methods brings one sharply to the realization that it is not known just what happens in the organism when these methods are applied and that reliable means of evaluating results are lacking. The indication seems clear that broad neurophysiologic investigations, including especially those in biochemistry, must be added to our psychologic approach. Only by such means may we hope to have some understanding of psychosomatic relations.

Successful Psychotherapy of a Choreic Syndrome: Report of a Case. DR. JOSEPHINE R. HILGARD and DR. S. A. SZUREK.

A 12½ year old child with chorea, but with no evidence of complications, was hospitalized because of the severe motor symptoms. Except for the electroencephalograms, the results of all laboratory tests, including temperature readings, the blood count, determination of the sedimentation rate, urinalysis, electrocardiographic studies, spinal puncture and roentgenographic examination of the chest, were without significance. The neurologist diagnosed the condition as Sydenham's chorea, and the electroencephalogram was said to be indicative of this condition.

Seven months after the onset of symptoms, direct psychotherapy was begun, and the motor symptoms disappeared within three weeks. At the same time, neurotic trends of perfectionism, indecision, worry and extreme ingratiation diminished.

The available data on the dynamics lead to the following formulation: The child was struggling with severely inhibited hostile impulses toward her family caused by many thwart-

ings, especially the parents' preference for a younger brother. The repression of the hostility seemed to be determined both by the mother's prohibition of any direct expression of it and by the mother's extreme self sacrifices for the children. The father's neurotic helplessness further contributed to the situation. The relief of the motor symptoms and the decrease in the acute neurotic trends coincided with the discussion of the patient's sense of guilt over her hostile wishes. The subsequent six months of treatment has seen a moderate, but incomplete, working through of some of her character trends. Treatment is still in progress.

DISCUSSION

DR. GEORGE J. MOHR: I am not familiar with cases of chorea in which treatment was carried out in this manner and under the particular conditions that obtained during the therapy in this case. The case as presented is incomplete, since the child is still under treatment and there are further factors relating to the family to be dealt with before Dr. Hilgard and Dr. Szurek are ready to present what they would consider a complete summary of the situation.

I am impressed with the fact that this child apparently did poorly in the ward for almost six weeks and that the generally permissive atmosphere of the ward in itself did not seem to be effective in helping her to deal with her symptoms. It seems that permissiveness on the part of the therapeutist, which was directed to the specific situations in which the child was unable to cope with the pressure of hostile feeling, expression of which had been prohibited by the overconscientious, moralistic mother, was effective in the relief of tension, with clearing up of the symptoms.

In evaluation of the process of recovery, the first emphasis should be placed on the exchange between the child and the therapeutist. The particular setting in which the therapy was carried out, however, was extremely important to this child. I should like to know whether the therapeutists feel they might have achieved a similar result on an ambulatory basis, that is, with the child coming in to see them and returning to her home. For this child the being out of a difficult family setting was of itself important, and the particular combination of ameliorative environment and specific relationship with the therapeutist operated well to produce rapid relief of symptoms. It would be of interest to see cases such as this one set alongside similar cases of children with ticlike disturbances, and perhaps epileptiform or other convulsive manifestations, in which probably a somewhat similar dynamic summary could be worked out.

DR. MAXWELL GITELSON: I wish to speak of the electroencephalographic studies, with reference to a possible "constitutional" factor in this case, which I think was overemphasized in the report, in contrast to the rather casually mentioned fact that the patient as an infant went through a series of alternate periods of feeding at the breast and at the bottle. There are not enough details to inform one of exactly what that meant, but one is justified in inferring that it represented an infancy situation of alternating gratification and tension. From what is known in general of the rearing of infants, such oscillations in the feeding experience speak for the probability that there has been established a pattern for a chronic attitude of expectant tension, which at this late date might well appear to be "constitutional."

A great deal of work has been reported on the electroencephalograms of children which purports to present evidence of "constitutional" disturbances in nearly all types of behavior disorders. To judge from much of this work, most child guidance clinics should be closed. Much of this investigation has shown no awareness or recognition of psychologic structure or dynamics. Often it fails to recognize the possibility that the electroencephalogram, like the respirogram, may be a manifestation of disturbed adaptation, rather than an index to its actual cause. One cannot speak with any clarity of "constitutional" influences until one knows more precisely what goes on during that earliest period of adaptation in which the structural and dynamic integration of the nervous system completes itself, while at the same time the child is establishing his primary interpersonal patterns. Is it not conceivable that the early reactions to the outer environment, expressing themselves through the immature nervous system, may have such a modifying effect on its rhythms that later chronic, but not "constitutional," disturbances may appear in the electroencephalogram?

Although a great deal of nominal attention has been paid to the period of infancy as being crucial to the ultimate pattern of the character and personality, much has yet to be learned about the psychosomatics of infancy and early childhood. It is in the period of primary psychobiologic adaptation and integration that one must search for the answers to many of the basic problems of psychosomatics, which are only approximately soluble in the adult.

I cannot conceive of a "constitutional" factor which, in response to environmental influences, would vary as much as has apparently occurred in this case, if one is to assume that the electroencephalogram is an index to the constitutional makeup. The constitution is a datum.

If the constitutional factor was primary in this case, while psychic factors were only secondary and served only to exacerbate the disorder in behavior, why should the electroencephalogram have varied as it did? Why does the "constitutionally" choreic substrate, as measured by the electroencephalogram, continue as a constant despite the improvement in behavior? Apparently, the patient became "constitutionally" nonchoreic when the environment made possible a more adequate adaptation and the symptoms disappeared.

It is my impression that this child showed evidence of chronic tension of environmental origin, of which nail biting was the chief manifestation. This tension seemed to have been kept under partial control by an obsessive-compulsive defensive system, which broke down under stresses described in the paper. The "choreic syndrome," then, appears to have been a new defense which permitted partial discharge of the tension in terms of actual motility and undoubtedly, also, by providing a symbolic equivalent for the specific affects concerned. I believe the child's improvement is the outcome of a diminution of tension under the conditions of a rational environmental and interpersonal experience and that this decrease of tension made her emergency defense unnecessary.

Dr. C. A. Neymann: Would it not have been more appropriate to entitle this paper "The Psychotherapy of an Anxiety Neurosis or a Hysteria, with Unilateral Choreiform Movements"? According to the history, which I may not have understood completely, the patient showed none of the classic symptoms which one associates with Sydenham's chorea. The authors speak of a choreic syndrome. This term seems to be somewhat misleading, since it suggests that the illness may have been Sydenham's chorea.

Sydenham's chorea is a definite, well established syndrome. Some years ago, in connection with artificial fever treatment, Dr. Blatt and I studied in detail 25 cases of Sydenham's chorea. Unilateral movements in Sydenham's chorea are rare. The authors did not mention heart murmurs, febrile reactions, changes in the sedimentation rate or other symptoms of rheumatic infection. It is doubtful whether a chorea which disappears in six weeks, even without rest in bed, does so because of any treatment that has been applied in the interim. In a large series of patients treated by means of artificial fever the average period of hospitalization was sixteen days and the longest thirty-nine days. If the patient of Dr. Hilgard and Dr. Szurek was suffering from unilateral Sydenham's chorea, which seems improbable, it is possible that time may in itself have brought about the cure of the disease. This could destroy, or at least cast a doubt on, the authors' premise that the excellent psychiatric treatment stopped the choreiform movements.

Dr. Alfred P. Solomon: I agree with Dr. Neymann as to the necessity of differentiating Sydenham's chorea, an infectious disease, sometimes complicated by endocarditis, from psychoneurotic motor tics. Textbooks on medicine do not distinguish well between the two conditions, even though they state that chorea sometimes spreads through the schoolroom as a habit spasm. This need for differentiation is emphasized because of the contraindications to ambulatory treatment of true infectious chorea. As one becomes more oriented psychosomatically, one must be increasingly alert to clinically closely simulated organic disease entities, which often demand an entirely different management.

The differential diagnosis is often difficult, particularly because psychologic considerations must be emphasized in the understanding and treatment of Sydenham's chorea. I believe the presence of true athetosis is the distinguishing motor feature of infectious chorea.

As to the case of Dr. Hilgard and Dr. Szurek, I agree with them that the permissive attitude of the therapeutist was of the greatest importance in securing the improvement. The hysterical motor component had a strong motivation of secondary gain, not only for attention, love and interest, but as a means of the child's presenting hostile impulses. Unless the need for the hostility is otherwise relieved, as it was in this patient, the tension will not subside. Here the kind, sympathetic, permissive attitude of the therapeutist relieved the tension of the rejection hostility. There were, of course, complicated psychodynamic factors, some of which have been brought out. Perhaps most important was the fact that the patient was approaching adolescence, more threatening in her case because insecurity and ambivalence about growing up were already present.

It is my opinion that in the practice of brief psychotherapy with such a patient as the one presented, the information secured from the patient and from other available sources, although it is important in making possible the formulation of the dynamics of the psychoneurosis, is of primary importance to the therapeutist for the purpose of determining the nature of the psychotherapeutic management, and that it is of much lesser, and only secondary, importance as a source of understanding and insight for the patient.

Dr. Franz Alexander: Independent of whether the present case was one of Sydenham's chorea or of some conversion symptom simulating chorea, I should like to ask the authors a question concerning the psychodynamics of the case. As I understand it, the psychodynamic

situation was as follows: The case was a classic example of the freudian formulation in so far as psychogenic factors were involved, with a classic neurotic symptom which substituted for a full fledged action. To put it simply, the patient had repressed certain hostile feelings, which therefore could not find legitimate outlets for expression. During the treatment, through a permissive technic, the patient was encouraged to express hostile impulses, and the symptoms accordingly disappeared. Now the same impulses found full expression through aggressive talk and behavior. I assume that the girl did not beat up those against whom she had hostility, but at least expressed it in some legitimate way which relieved the tension. In listening to the paper, one might have received the impression that the therapeutists felt that the problem of treatment in this case could be solved by simply encouraging the patient to get rid of the pent-up hostilities through open expression of them. I think, however, that the real problem is to make the patient overcome her hostile impulses by getting deeper insight into the motivations behind them. Then the therapy will lead to permanent improvement. To overcome the hostile impulses is the real therapeutic aim, and not alone to help the patient to abreact them. I must assume that, apart from this freedom of expression of hostilities, the patient learned something about the background of her hostile impulses; otherwise, the response was merely an abreaction, which, as is known, does not result in a lasting cure. If this girl merely abreacted her hostilities through encouragement, the hostilities will generate again, and some form of neurotic trouble will recur. In this case the patient may have been cured of Sydenham's chorea, or of some choreiform syndrome, but sooner or later she will have other trouble. Sometimes—fortunately seldom—psychoanalysis cures a patient of his symptoms by converting them into a character defect. Those unacceptable impulses, which before treatment the patient expressed in harmless symptoms—at least harmless for the environment—he afterward expresses in behavior, in human interaction. The therapy is obviously not finished at this point. Simply to encourage people to give vent to their formerly repressed hostile impulses is not good service. I do not think that is what happened in the case of this girl. It was said that she became more popular in the ward. This could not be the result of uninhibited aggressive behavior. I should like the authors to tell a little more about what took place in this patient's personality.

DR. S. A. SZUREK: We cannot answer Dr. Mohr's question. We have not had any experience with ambulatory treatment. In my opinion, some less intensive therapy with concomitant collaborative treatment of the parent would have to be carried out. Then it might be possible over a longer period to achieve some effects. I agree with Dr. Mohr that hospitalization in the ward, so that the child was removed from the immediate situation, made the psychotherapy easier.

With regard to Dr. Neymann's comments on chorea, we are not under the impression that chorea is always associated with signs of infection. Further, in this particular case, the neurologist whom we consulted first recommended sedation and isolation and then discussed with us the indications for fever therapy. If the condition was not chorea, it certainly looked like it. I don't think the choreiform movements were a simple hysterical symptom, at least it was not easy to make such an assumption prior to the institution of psychotherapy.

I am not sure I can accept Dr. Alexander's suggestion that relief of the motor symptom was the result of mere abreaction and that there was no change in character, for I have the impression that both the influences of the ward and the psychotherapy have not been merely in the nature of catharsis and abreaction. Definite changes in the girl's character are going on. This child has really changed; in general appearance she is much prettier, and this is not due alone to her having her teeth fixed and getting good glasses.

As to Dr. Gitelson's comment, I might state my own position with regard to "constitution" as follows: Some changes in the central nervous system which occur as the result of the earliest conditioning experiences of childhood become part of the constitutional factor, inasmuch as they are not easily modifiable, or may not be modifiable at all, by later interpersonal influences. In this sense, one could differentiate in the constitution of the individual organism between the congenital and hereditary factors and the changes induced during the extremely plastic and modifiable phase of development of the central nervous system shortly after birth.

The electroencephalographic changes are a matter for further study. Although we experienced some difficulty with our own electroencephalographer in defining terms and establishing a basis of understanding, it should be possible for the electroencephalographer and the psychiatrist to understand one another. We have had other experiences in which the electroencephalographer suggested medication on the basis of his recordings (notably, in the case of a psychotic child of 4 years, who later recovered) and in which we did not agree with the recommendation and noted both clinical improvement and changes in the electroencephalogram with continued psychotherapeutic effort, but without medication. It is, I am certain, a field for further collaborative study.

Schizophrenia: Structural Analysis and Metrazol Treatment. DR. KURT EISSLER.

The case histories of 5 schizophrenic patients who received psychotherapy and those of 3 patients treated with metrazol are briefly presented. The psychotherapeutic method applied was that of psychoanalysis adapted to the individual patient. Of the 3 patients treated with metrazol, 2 had schizophrenia, and the third received the treatment because of acute anxiety. All 3 patients showed signs of dementia when examined some years after the treatment.

It is suggested that metrazol treatment be employed only after it has been determined that psychotherapy will be unsuccessful in the particular case.

DISCUSSION

DR. LADISLAS MEDUNA: Dr. Eissler's reasoning can be summarized as follows: He divides schizophrenia into two types: the so-called schizoid psychosis and true schizophrenia. The schizoid psychosis corresponds in many respects to psychogenic schizophrenia, while true schizophrenia, whatever it may be, must be considered the result of a disorder in the function of the nerve cells of the brain.

Dr. Eissler states that in many cases even the most thorough structural analysis cannot establish differences between the symptoms of the schizoid psychosis and those of a chronic schizophrenic disorder. Nevertheless, he adds, the schizoid psychosis is amenable to psychoanalysis. If the word psychoanalysis is replaced by the broader term psychotherapy, I believe every one will agree with Dr. Eissler.

The difficulty begins when this general statement is applied to an individual case. According to Dr. Eissler: "Psychotherapy adapted to the individual requirements of the patient is preferable to pharmacologic treatment." This statement is too dogmatic to be true. Dr. Eissler should have stated only that in cases of psychogenic schizophrenia psychotherapy is to be tried first. But even this statement has only a limited value, for there are psychogenic schizoid reactions which develop into a true schizophrenic process, and, on the other hand, there are cases of true schizophrenia in which a psychologic superstructure entirely covers the underlying pathologic process.

Dr. Eissler's main objections to the convulsion treatment are indicated in the following quotation: "All 3 patients had one feature in common, a decrease in their capacity for self observation; all 3 patients treated with metrazol showed in addition lack of emotional depth and withdrawal from personal contacts. . . . It appears that these patients showed signs of a syndrome which may be classified as dementia. Such a syndrome is not the usual massive picture associated with this term, but a peculiar new form . . . which induces an automaton-like existence."

I refuse to accept these symptoms as the consequence of metrazol treatment, because they are, according to Kraepelin, the fundamental symptoms of dementia praecox. "This peculiar and fundamental want of any strong feeling of the impressions of life, with unimpaired ability to understand and to remember, is really the diagnostic symptoms of dementia praecox. . . . The peculiar feeblemindedness seems to be decisive of the diagnosis." This dementia is characterized, according to Kraepelin, by emotional dulness, loss of mental activity and signs of automatic obedience. These profound changes in the character with loss of mental and emotional feeling, absence of any understanding of the past illness and want of freedom in action and behavior, persist even during spontaneous remission, as he observed about forty years ago.

I have no doubt that Dr. Eissler's observations are identical with those of Kraepelin; consequently, the "peculiar dementia" he described is the fundamental symptom, and in most cases the irreversible consequence, of the schizophrenic process, and not the result of shock treatment.

CHICAGO NEUROLOGICAL SOCIETY AND ILLINOIS PSYCHIATRIC SOCIETY

Joint Meeting, Dec. 17, 1942

FRANCIS J. GERTY, M.D., *President of the Illinois Psychiatric Society, in the Chair*

Discharged Veterans of World War II, in State Hospitals and in the Community. DR. CONRAD S. SOMMER and DR. JACK WEINBERG.

The rehabilitation of the neuropsychiatrically disabled veteran of World War II is already presenting a problem, which undoubtedly will grow in proportion to the increase in returned men. To avoid the mistakes of the past, and to understand the problem of rehabilitation more

thoroughly, we undertook a study to acquaint ourselves with the caliber of the returned men, their needs, aspirations and expectations. The survey also attempted to evaluate the neurotic drives of these patients and the best method of their utilization for the common good.

By Aug. 1, 1942 2,354 veterans of World War II had been returned to Illinois. Of this number, 1,014, or 40 per cent, returned because of neuropsychiatric and personality defects. A sampling of these patients, made by a psychiatric and recreational rehabilitation bureau, which visited the homes of 75 veterans, questioned 300 of them and interviewed 38 of a selected group, revealed that 10 per cent, or 101 veterans, were in state institutions for the mentally ill, while 90 per cent were living in the community. The vast majority, 86.7 per cent of those in the community, were employed, and a substantial majority appeared to be making a fair adjustment. The abundant opportunities for employment, the pressure applied to every one to work and to produce and the widely felt urge to contribute to the war effort are no doubt interfering wholesomely with the temptation of many of these veterans to yield to neurotic dependence.

Of the difficulties encountered, the most serious was the phraseology of the discharge certificate: "undesirable traits and character"; "inapt"; or "mental condition," which seemed to stigmatize the man and, in many instances, interfered with his obtaining adequate employment.

We recommend the following program:

1. A medical program to meet the returned veteran's needs, as indicated by the physical disability. Medical men should avoid undertreatment or overtreatment of these patients, as the former breeds discontent while the latter invites hypochondriasis and invalidism. Psychiatrists should participate in the medical therapy as hygienists.

2. The provision of needed psychiatric institutional treatment, not to be unduly prolonged. This should be facilitated by revision of commitment laws, which, through stigmatization, often prevent or delay the returned men from seeking help in institutions for the mentally ill.

3. Short periods of extramural psychotherapy aimed at social and occupational adjustment for the veteran, which would lead to his becoming self sufficient.

4. Consultant advice to groups working intimately with the rehabilitation of veterans, such as the American Legion, the American Red Cross, the Veterans' Administration, the State Veterans' Divisions and bureaus of vocational rehabilitation. (A useful by-product of such activities at this time in Illinois is the opening of facilities in bureaus of vocational rehabilitation to patients with mental disorders, who formerly were excluded.)

5. Some form of certification for those veterans on whom the discharge certificate from military service casts an unnecessary stigma, interfering with vocational adjustment.

6. Further cooperation and mutual endeavor of psychiatrists and veterans' organizations to develop among veterans and psychiatrists the conception that the best reward a nation can offer its returned veterans is the development of ability to work and be independent.

7. Active efforts on the part of psychiatrists in helping veterans' organizations to formulate new laws which would avoid the errors of the past, providing a justly recompensing bonus to all veterans and, in addition, setting up a program which would insure rapid rehabilitation and return to employment of the maximum number of disabled veterans and thus reduce the amount of neurotically induced invalidism.

Psychiatrists should not take the nihilistic attitude that there will be a vast amount of neurotic dependence about which nothing can be done. The actual amount of lifelong neurotic invalidism will be greatly influenced by an efficient program for rehabilitation of discharged veterans of World War II.

DISCUSSION

DR. ALFRED P. SOLOMON: This paper confirms a recently reported study I made of 54 candidates rejected by the induction board for psychiatric reasons. As with the patients of Dr. Weinberg and Dr. Sommer, all but 4 had secured employment. Such observations are of value in determining the reversible character of personality reactions to a given standardized disagreeable situation from which one has been removed.

If I understand the authors correctly, I differ with regard to one of their recommendations. I do not believe that persons who are making even a fair industrial adjustment should be given psychiatric treatment so long as they continue at their employment. A continuing work adjustment, with the present high wages and patriotic implications, is in itself a progressive form of psychotherapy, leading to rehabilitation more successfully than any other method.

DR. RALPH HAMILL: I do not recall that the essayists mentioned the tendency of these discharged men to claim a physical disability on which to base their complaint. My experience in the last war was that nervousness was the last thing of which they complained; whether it will be the same in this war I do not know, but with the scientific recognition of nervousness many men are beginning to say that they cannot sleep because of nervousness, and that therefore

they cannot work. I wonder whether the physical side of medicine is going "to be wise to" the neurotic side, or whether it will focus attention on something physical not of great importance and so furnish the veteran with a basis for complaint.

I am much concerned about the accessibility to the neuroses of the public at large. Whether we of the scientific side of life are giving justification to men to complain of nervousness, and so are suggesting disability, is a matter of much concern, to my mind. If the physicians are known to recognize a neurosis, that makes it good.

For example, men blamed gassing and influenza for their inability to settle down after the last war. Both these conditions were "known" by the medical profession to predispose to pulmonary tuberculosis. Any breathlessness, cough or feeling of "closeness," therefore, was worked up by the patient as a threat of tuberculosis, so that he could not return to his inside job at a desk or counter, or in a shop.

DR. FRANCIS J. GERTY: If I understood correctly, only 2 of the total group have so far talked about compensation. I hope the figure will remain as low as that.

DR. CONRAD SOMMER: The proportion of veterans asking for disability compensation was 2, out of a sample of 75 men, an unusually small percentage.

We are not in agreement with Dr. Solomon's point that psychotherapy is usually not indicated for men who are well adjusted in their work. In a considerable number of cases work adjustment is precarious because of poor mental health, and is likely to become progressively poorer unless some psychotherapy and redirection are given.

In response to Dr. Hamill's question, there has been a definite change in that physicians in military services make neuropsychiatric diagnoses much more freely than in the last war and do not hesitate to inform the patient that the cause of his discharge is of a mental nature. This, of course, does not prevent the veteran from looking for physical reasons for his "nervous" condition. He has, however, some understanding of the mental nature of his disability.

We propose the following form for the certificate of rehabilitation:

CERTIFICATE OF VOCATIONAL FITNESS

Date.....

This certifies that....., having been discharged from military service because of a disability interfering with his successfully performing military duties, has been examined by the undersigned and has been found to be fit for civilian employment.

.....M.D.

In general our observations were quite unexpected to us. The first veteran I visited in his home was a Negro 22 years of age, who was asleep in bed at 10 in the morning. My snap diagnosis was that here was a neurotic person, sleeping the working hours away, expecting to be supported by his government. How erroneous my preconceived notion was became apparent when he told me of his employment in a defense factory at night. The reason for his military discharge was psychoneurosis. This veteran, and many others whom my colleagues and I have contacted, demonstrated clearly that a proper program of rehabilitation and an appropriate work situation can prevent latent neurotic dependence from developing into neurotic invalidism, requiring hospitalization and government support in the majority of cases.

Chronic Tuberculous Meningoencephalitis. DR. JOHN R. GREEN.

This presentation concerns a man aged 23 who had suffered from major epileptic seizures between the ages of 10 and 20 years, diabetes insipidus after the age of 20 and jacksonian seizures of sensory type for seven months prior to his admission to the hospital. Ventriculograms demonstrated dilated ventricles and poor filling of the third ventricle. He was considered, until his death, to harbor a neoplasm in the region of the hypothalamus.

Necropsy revealed leptomeningeal nodules which appeared tuberculous. In addition, a healed childhood type of tuberculosis of the lungs and a few caseous and calcified mesenteric lymph nodes were encountered. Microscopic examination revealed widespread granulomatous changes in the lungs, liver, spleen, mesenteric lymph glands, leptomeninges and neural parenchyma, including the neurohypophysis. The majority of the lesions were noncaseating epithelioid tubercles, similar to those of sarcoidosis. In addition, endarteritis and other inflammatory changes were noted in the brain. Caseation necrosis had completely destroyed several mesenteric lymph glands and was also apparent in a number of the tubercles in the nervous system.

That tuberculous leptomeningitis may pursue a long chronic course is emphasized, such a condition usually being confused with tumor of the brain and the tubercle bacillus rarely being demonstrated. Sarcoidosis is discussed. The prevailing evidence, including the observations in this case, seems to indicate that sarcoidosis represents a chronic proliferative phase of tuberculosis.

DISCUSSION

DR. GEORGE B. HASSIN: The changes that Dr. Green so well described in his case remind me of a similar condition in a case of tuberculosis of the spinal cord (Thalhimer, W., and Hassin, G. H.: *J. Nerv. & Ment Dis.* **55**:161 [March] 1922). There were the same hyperplastic phenomena in the pia-arachnoid, with cellular infiltrations and vascular changes, but they were associated with pachymeningitis. In the cord two solitary tubercles could be discerned—one in a state of fibrosis and the other in process of development. The meningeal changes were especially noticeable at the level of the tubercles and were considered secondary to involvement of the spinal cord. A similar condition was evidently present in Dr. Green's case. His patient exhibited for several years definite signs of involvement of the brain, as evidenced by diabetes insipidus and the changes in the pituitary body seen at autopsy. The cerebral changes evidently affected the meninges, which, like the changes in the spinal meninges in my case, were chronic and secondary. The presence of healed tuberculosis in one of the lungs also suggests that the meningeal changes were tuberculous, and not those of sarcoidosis. It is doubtful whether there is on record a bona fide case of sarcoidosis of the central nervous system without lesions elsewhere in the body.

DR. BEN W. LICHTENSTEIN: I have little to add to Dr. Hassin's discussion of Dr. Green's presentation. I saw the microscopic preparations of the original biopsy specimen, and the granulomatous character of the lesion was the outstanding feature. A diagnosis of tuberculosis, however, was not made at biopsy. I had the opportunity of studying the preparations made from the autopsy material, and these were definitely tuberculous. One's conception of the microscopic features of tuberculosis is dependent on one's experience. The pathologist who examines biopsy specimens from enlarged lymph nodes appreciates how pleomorphic the lesions of tuberculosis can be, varying from foci of specific inflammation, with caseation and Langhans' giant cells, through foci of nonspecific inflammation to lesions in which the inflammatory reaction is minimal, the lesions being almost entirely a manifestation of reticulum proliferation, as in the case of sarcoidosis.

This may not be the proper place for a discussion of sarcoidosis, for pathologists themselves are far from agreement on the exact relationship of sarcoidosis and tuberculosis. Dr. Green's case was of unusual interest because it was an instance of chronic tuberculous meningoencephalitis which clinically resembled a cerebral neoplasm.

Electroencephalographic Study of the Local Effects of Electric Shock: A Preliminary Report. DR. LOUIS D. BOSHES, DR. CHESTER W. DARROW, DR. ALFRED P. SOLOMON and DR. JULIAN H. PATHMAN.

Electroencephalographic activity immediately beneath the point of application of the shocking electrodes showed initial increase in the potential, decreased frequency and decreased blocking of the alpha waves. With continuation of treatment the electroencephalogram progressively acquired the characteristics of interseizure records. To determine local effects we have departed from the conventional placement of the shocking electrodes in favor of an asymmetric arrangement, such as the left frontal and the right occipital area. By this means it has been possible to compare electroencephalograms from shocked and those from unshocked areas. The results suggest impairment of cerebral metabolism as a factor in the improvement following shock therapy.

DISCUSSION

DR. R. W. GERARD: The authors are obviously to be congratulated on the ingenious and simple device they have introduced for comparing shocked and unshocked symmetric parts of the brain. I should have been dubious about its working so well because, owing to the resistance of the scalp and the skull, the shock undoubtedly spreads more widely than to the region directly underneath the electrodes. Perhaps this physical spread may have some bearing on the gradual spread of the changes in the electroencephalogram. Is it possible that the later changes seen in the unshocked occipital lobe are not wholly due to neurogenic spread from the shocked region, but are partly to be explained by the spread of the shocking current itself?

It also occurred to me that in every case shown in which the shock produced an increased symmetry, the shocked side was initially of lower amplitude. If the more active side receives the shock, is the asymmetry increased?

One would undoubtedly have to agree with the main conclusions of the authors, namely, that parallelism in the electric changes in the brain and in the clinical cause of psychosis is evidence of some relation between cerebral physiology or pathology and the mental manifestations, of some "organic" tie-up. I am rather more reserved in accepting some of the particular interpretations of the mechanism of action of the shock treatment. It is suggested that the observed effects—increased amplitude and decreased frequency, decreased blocking and reversibly

increased irregularity—are evidences of a decrease in cerebral metabolism. There are reasons which make me rather suspicious of so simple a picture. The "mechanical" side of the picture is emphasized, namely, that with slower frequency there is more time for amplitude to manifest itself. That relationship is not of itself evidence of a metabolic change, but rather is indicative of change in the "trip" mechanism to the nerve cell, as Libet and I have called it. Further, when frequency and amplitude do change on this basis, they are reasonably parallel quantitatively—a halving of the amplitude for a doubling of the frequency. I do not believe the records showed this. In all cases effects on amplitude were greater than those on frequency.

When changes of amplitude occur independent of frequency, or even take place in the same direction, they cannot be due to a simple trip mechanism; they are due, I believe, to changes in metabolism. An increased amplitude at a given frequency is evidence of an increase, rather than a decrease, in metabolism. In the first slide there was a striking increase in amplitude and no change in frequency, which I should consider evidence of increased, not decreased, metabolism of the brain. Frequency can be increased without change of amplitude, or both may increase together, as with a rise in temperature. Perhaps I am a little prejudiced in my point of view, since some years ago in the ARCHIVES I invited criticism by interpreting all shock and related treatments of schizophrenia as acting to increase the metabolism of the brain. Thyroid, which has been reported to have a good effect, certainly increases the metabolism of the brain. The other treatments which give an immediate depression, such as metrazol shock, perhaps produce secondary enhancement of oxidation, such as occurs after anoxia itself.

However, it may be the other way. Two effects are reported which suggest diminished cerebral activity: pronounced irregularities which appear and then disappear and the decreased blocking. The latter would result if the cells were less irritable to incoming impulses or if impulses were more or less blocked. Further, extended hypoglycemia decreases cerebral metabolism while it exists (whatever happens later) and acts from the higher centers of the central nervous system down the neuraxis. Positive therapeutic results in such cases, as after leukotomy, might thus be due to decreased activity of higher centers, or release of lower ones, as well as to increased activity of higher centers, as previously argued.

I am sure the authors do not feel their evidence is conclusive. The important thing is that there are changes in brain waves which are correlated with the mental condition; further studies will show what the correlation is. But whether shock therapy acts by adding some positive factor or by taking away some negative one, I do not think is yet known.

DR. ALFRED P. SOLOMON: Dr. Gerard's interpretation of our observations may be correct, but the outstanding fact is that easily demonstrable electroencephalographic changes during electric shock have been observed to be reversible. When these physiologic changes are correlated with associated clinical observations, it is possible that the implications will afford an important contribution to the understanding of shock therapy. Such a study is in progress and will be presented in a later communication.

DR. VICTOR E. GONDA: I should like to have some information on the application of the electrodes in the frontal and occipital regions. The shaving of the skull before application of the electrodes was complete, I assume. Was a stronger current or a longer time needed for development of the convulsive seizure than with the usual bitemporal application of electrodes? I am also anxious to know whether, during this interesting work, some different pattern of the seizures was ever observed, as the electrodes were placed over different parts of the skull.

In their conclusions the authors mentioned fear reactions of the patients. This is surprising with electric shock, since such undesirable complications can and should be eliminated.

It is a satisfaction to hear that fifteen or more treatments were used, because lately I have noted that in many institutions the treatment is stopped before an adequate number is given. I wish also to emphasize with the authors the importance of psychotherapy in connection with shock treatments. Psychotherapy is at least as important as the convulsive seizure if satisfactory results are to be obtained.

DR. LOUIS D. BOSHES: The areas used were circularly shaved for about 2 cm., just enough to get the electrodes in place; they were about the size of a penny. We made our own electrodes and clasps. There was no increase in the amount of current used, and considerably less was necessary in some cases to achieve a good reaction.

Nothing definite was noted as to the pattern of the shock. Sometimes a severe reaction was obtained with a small amount of current, as well as with larger amounts. We did not notice that the shock reaction originated in any particular area. As to the number of treatments, some patients were given as many as twelve or fifteen. We stopped treatment of 1 patient when he began to have disturbed patterns, after his seventeenth treatment.

DR. CHESTER W. DARROW: I do not question the possibility suggested by Dr. Gerard that the observed spread of effects from the shocked to the unshocked areas may be due in part to the spread of current. However, the fact that spread of dysrhythmia becomes prominent only after repeated treatments suggests neurogenic, rather than merely physical, factors.

In anticipation of the frequently observed impairment of the alpha potential by convulsants, we early in our work applied the shock to the side with the higher, more normal, occipital alpha potential. After we found an initial local improvement of alpha potential in the shocked area, we consistently applied the shock to the less normal side.

Dr. Gerard called attention to something I had not previously noted. In the first slide there was an increased amplitude of potential in the shocked area without an appreciable decrease in frequency. This would ordinarily be interpreted as indicating an increase in metabolic activity. In other cases there was consistently a decrease in frequency whenever there was an increase in amplitude, and often even when the amplitude was not increased. That alteration in frequency may be due to change in the "trip" mechanism controlling repetitive discharges, as suggested by Dr. Gerard, is quite possible. As already pointed out, we believe that the more immediate results of electric shock, both physiologic and psychologic, may be accounted for by a decrease in cerebral metabolism. Whether or not there may later be a "rebound" with a subsequent increase in metabolism, our data do not answer conclusively.